Report of the Independent Fact-Finding Panel on Herbicide 2,4,5-T

Final Report

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Leonard Ritter PhD, Fellow, Academy of Toxicological Sciences, Panel Chair

Aaron Blair PhD, Fellow, American College of Epidemiology, Ramazzini Collegium, American Epidemiological Society

Nancy I. Kerkvliet PhD

Elliot A. Sigal HBSc

Jeanne Mager Stellman PhD

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Report at a glance

Why this report?

Beginning in the late 1940s, the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), a synthetic plant growth hormone, was used widely for brush control in Ontario, Canada, and worldwide. Like all pesticides, this herbicide contained an active ingredient—2,4,5-T—along with other chemicals, such as solvents and emulsifiers. Due to poorly controlled manufacturing conditions, this herbicide could also contain inadvertent contaminants, with 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) causing the most concern. Much of the worldwide debate on 2,4,5-T's health effects has focused on TCDD.

By the early 1970s, the Government of Canada was aware of concerns about possible health effects from exposure to 2,4,5-T and its contaminants. By 1979, 2,4,5-T use was suspended in Ontario, 6 years before it was deregistered by the federal government.

In 2011, in response to public concern about possible long-term health effects following 2,4,5-T exposure, the Government of Ontario formed an independent panel to review use of this herbicide in the province and to evaluate possible health effects.

What were the review's goal and scope?

In March 2011, the Ontario government established an independent fact-finding panel, and appointed a chair. The chair assembled an independent panel of experts in epidemiology, toxicology and exposure and risk assessment from across North America (http://ontarioindependentfact-findingpanelon245t.ca/). The panel's goal was to assess potential exposure and any risks that Ontario government use of 2,4,5-T could pose to population health.

More specifically, the panel was charged with determining:

- Where, when, and how much 2,4,5-T government ministries and agencies used in Ontario
- How 2,4,5-T was used in Ontario and whether exposure to 2,4,5-T could affect human health, including that of workers and bystanders
- How 2,4,5-T was prepared, applied, and stored, as well as what provincial occupational health and safety practices, and laws, standards, and workplace practices were in place during the period of use in Ontario

The panel's mandate focused on 2,4,5-T use by provincial government departments and agencies. It did not cover other widely used herbicides, such as 2,4-dichlorophenoxyacetic acid (2,4-D) and 2 (2,4,5-trichlorophenoxy) propionic acid (2,4,5-TP), or use by others, such as homeowners or municipalities.

How was the review conducted?

To find out where, when, and how much 2,4,5-T was used in Ontario as well as how it was prepared, stored, and applied, the panel reviewed approximately 4700 records from government ministry and agency files. Interestingly, Ontario government departments and agencies were not the primary users of 2,4,5-T in Ontario; based on available records, private users and municipalities used far more of this herbicide.

To assess whether 2,4,5-T exposure could affect human health, the panel relied on basic information from research publications, reports from recognized international agencies such as the United Nations World Health Organization, and reports published by international, federal, and provincial government departments, such as the U.S. Environmental Protection Agency and Health Canada.

To characterize the potential for 2,4,5-T and TCDD to adversely affect human health, the panel adopted an internationally accepted approach for establishing safe exposure levels. Below this threshold, or safe level, adverse effects are not expected even with daily exposure for a lifetime.

The international consensus from animal and human studies is that 2,4,5-T, on its own, has only limited toxicity, while its contaminant, TCDD, is toxic and is a possible cause of cancer and other adverse outcomes in people. For the TCDD part of the risk assessment, the panel used *tolerable daily intake* levels proposed by the United Nations Joint FAO/WHO Expert Committee on Food Additives and also accepted by the Canadian and Ontario governments as the threshold, or "safe" exposure level. For the risk assessment of 2,4,5-T, the panel used a similar approach, noting that because of its more rapid breakdown in the human body and in the environment compared to TCDD, exposures to 2,4,5-T would generally be short-term in duration. In contrast, because TCDD breaks down very slowly, exposures to TCDD were considered to be of a long-term nature.

To conduct the exposure assessment, the panel developed exposure scenarios for the three primary uses of 2,4,5-T by government departments and agencies:

1. brush control in forests by the Ministry of Natural Resources (MNR)
2. rights-of-way clearance around power lines by Ontario Hydro
3. weed and brush removal along major roadways by Ministry of Transportation (MTO)

These scenarios were used to model possible human exposures to 2,4,5-T through direct inhalation, skin contact, and indirect exposure through ingestion of contaminated berries, fish, wild game, plants, or soil.

People who were considered at risk of exposure included those who worked with 2,4,5-T, those who lived near areas that were treated, and those who recreated in or near sprayed areas. However, since no specific human exposure assessment was done when 2,4,5-T was used, and because it was used many years ago, the estimates developed by the panel involved some uncertainties and many assumptions. To ensure the assessment captured the range of possibilities, the panel estimated low, central, and high exposures for each scenario and assessed risk by determining a *margin of safety* for various population subgroups. A *margin of safety* approach allows one to compare exposure levels determined to be "safe" from published studies with estimated exposures in the Ontario populations studied by the panel. The panel's exposure assessment included assumptions that erred on the side of caution to ensure they did not underestimate disease risks.

What were the panel's findings?

Based on some of the exposure assessment scenarios, exposure substantially exceeded safe thresholds in some subgroups of the Ontario population. These scenarios modelled occupational exposures where Ontario Hydro, MNR, and MTO workers were repeatedly exposed, over long periods, to the mid and highest levels of TCDD. Such scenarios would represent workers who were involved in mixing, loading, and applying herbicides and were exposed regularly to TCDD-contaminated 2,4,5-T for long periods. However, the toxicological reference values (TRVs) used to predict safe thresholds of exposure are required by regulatory policy to err on the side of safety. Thus, even when exposure estimates exceed safe threshold levels, a wide margin of safety is incorporated into the estimates and adverse health effects may not occur. The risk assessment only indicates that acceptable margins of safety have been exceeded for certain occupationally exposed groups, and that their health could have been affected.

Bystander exposures, defined by the panel as being of a brief nature and not from personal direct use of the herbicide, exceeded a safe threshold only in the case of Ontario Hydro and MNR and only for the highest TCDD exposure scenario, and the safe threshold was only marginally exceeded. No other bystander exposures were found to exceed the safe threshold. None of the bystander exposures to 2,4,5-T for Ontario Hydro, MNR, and MTO exceeded what was considered to be a safe threshold of exposure. The panel concluded that the risk of developing a disease due to bystander exposure to 2,4,5-T/TCDD would be very low.

It is important to note that the panel conducted a population-level assessment, which characterizes risk for groups of individuals. Such assessments, however, cannot be used to determine whether or not an exposed individual will actually develop a disease or adverse health event from exposure to 2,4,5-T or TCDD.

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List of acronyms

2,4,5-T = 2,4,5-trichlorophenoxyacetic acid

2,4,5-TP = 2-(2,4,5-trichlorophenoxy) propionic acid

2,4-D = 2,4-dichlorophenoxyacetic acid

ADI = acceptable daily intake

AHETF = Agricultural Handler Exposure Task Force

AhR = aryl hydrocarbon receptor

BASF = biota-to-sediment accumulation factor

BTF = bio-transfer factor

BW = body weight

DLC = dioxin-like chemicals

IARC = International Agency for Research on Cancer

IOM = Institute of Medicine

IRIS = Integrated Risk Information System

LOAEL = lowest observed adverse effect level

MCPP = methylchlorophenoxyproprionic acid

MET = Ministry of Education and Training

MNR = Ministry of Natural Resources

MOE = Ministry of Environment

MTO = Ministry of Transportation

NAS = National Academy of Sciences

NIOSH = National Institute for Occupational Safety and Health

NOAEL = no observed adverse effect level

NPC = Niagara Parks Commission

NTP = National Toxicology Program

OMAFRA = Ontario Ministry of Agriculture, Foods and Rural Affairs

OPAC = Ontario Pesticides Advisory Committee

ORETF = Outdoor Residential Exposure Task Force

PCB = polychlorinated biphenyls

PCDD = polychlorinated dibenzodioxins

PCDF = polychlorinated dibenzofurans

PCPA = Pest Control Products Act

PHED = Pesticide Handler Exposure Database

PMRA = Pest Management Regulatory Agency

PPT = parts per trillion

RAF = relative absorption factor

RfD = reference dose

RR = relative risk

SMR = standardized mortality ratio

TCA = trichloroacetic acid

TCDD = 2,3,7,8-tetrachlorodibenzo-*p*-dioxin

TDI = tolerable daily intake

TEF = toxic equivalency factor

TEQ = total toxic equivalent

TRV = toxicological reference value

UE = unit exposure

U.S. EPA = United States Environmental Protection Agency

WHO = World Health Organization

Glossary[1]

Acceptable daily intake (ADI): Estimate of the amount of a foreign substance (e.g., food additive) in food or drinking water that can be ingested daily over a lifetime without appreciable health risk.

Acid equivalent (a.e.): The portion of a formulation (e.g., ester, salt, or amine) that could be converted back to the parent acid.

Active ingredient (a.i.): The chemical in a mixture responsible for its biological effects.

Acute exposure: Short-term exposure.

Acute toxicity: 1. Adverse effects occurring (a) up to 14 days after a subject is given a single dose or exposed to a given concentration of a test substance or (b) after multiple doses (exposures), usually within 24 hours of a starting point, which could be exposure to the toxicant, loss of reserve capacity, developmental change, etc. 2. Ability of a substance to cause adverse effects within a short time of dosing/exposure.

Aerial spray: Spraying from an aircraft.

Application rate: The quantity (mass, volume, or thickness) of material applied per unit area.

Aryl hydrocarbon receptor (AhR): An intracellular protein that binds aryl hydrocarbon compounds such as TCDD and is responsible for mediating the toxicity of such compounds.

Bioaccumulation: Progressive increase in the amount of a substance in an organism/part of an organism; occurs when the organism takes in the substance faster than it can remove/excrete it.

Bioassay: Procedure for estimating the concentration or biological activity of a substance by measuring its effect on a living system compared with the effect of a standard substance.

Bioavailability: The fraction of a substance to which an organism has been exposed that is available to the target tissue.

Biomarker: Indicator that signals an event or condition in a biological system or sample and gives a measure of exposure, effect, or susceptibility; may be chemical, biochemical, physiological, behavioural, etc.

Canopy intercept fraction: The amount of precipitation intercepted and then evaporated from a forest canopy.

Carcinogenicity: The ability of a chemical, physical, or biological agent to cause cancer.

Carrier: One of several non-active components of a pesticide formulation (including herbicides); other components typically include a *solvent* and an *emulsifier.*

Chronic exposure: 1. Long-term exposure. 2. Ongoing exposure(s) over (a) an extended period or (b) a significant proportion of the test species', group of individuals', or population's lifetime.

Chronic toxicity: 1. Long-term toxicity. 2. Adverse effects following chronic exposure to a substance.

Contaminant: Impurity present in a substance.

Dermal absorption: Absorption of a substance through the skin.

[1]This glossary was prepared using commonly defined terms accepted by the Independent Fact-Finding Panel on Herbicide 2,4,5-T, as well as established sources such as the International Union of Pure and Applied Chemistry (Duffus, J.H. , Nordberg, M. and Templeton, D.M. 2007. Glossary of terms used in toxicology, second edition. Pure and Applied Chemistry 79, 1153-1344).

Dioxin-like chemicals: A family of toxic chemicals that have a similar structure and cause harm through a similar mechanism; examples include polychlorinated biphenyls (PCBs), polychlorinated dibenzo-*p*-dioxins (PCDDs), and polychlorinated dibenzofurans (PCDFs).

Dose-response assessment: 1. Assessment of the association between the amount of chemical absorbed by the body (the *dose*) and incidence of a defined biological effect in an exposed population, usually expressed as percentage. 2. In exposure assessment, the process of determining the relationship between the stressor dose and a specific biological response.

Drift loss fraction: The fraction of applied airborne liquid or solid that moves away from the target area during application.

Efficacy: A pesticide's ability to fulfill the claims made on its label, including the extent of pest control and adverse effects on the host, i.e., toxicity to animals and plants.

Embryotoxicity: Ability of a substance to harm an embryo (between conception and the fetal stage), including malformations and variations, malfunctions, growth changes, prenatal death, and functional changes after birth.

Endocrine: Having to do with hormones or the glands that secrete hormones into the bloodstream.

Endogenous: Produced or caused by materials within an organism (for example, hormones).

Epidemiology: The study of disease in human populations; often involves comparing two or more groups of people who are as alike as possible, except for the factors being investigated.

Epigenome: The set of chemical compounds that control how an organism's genes are expressed by turning various genes on or off at different points in time; determines how a cell develops and what it produces.

Exogenous: Caused by or derived from materials outside an organism (for example, chemicals in the environment).

Exposure: 1. Any contact with a substance by swallowing, breathing, or direct contact (such as through the skin or eyes); may be either short term (*acute*) or long term (*chronic*). 2. Process by which a substance becomes available for absorption by the target population, organism, organ, tissue, or cell—by any route.

Exposure pathway: The physical course a chemical or pollutant takes from the source to the exposed organism.

Fetotoxic: Harmful to a fetus, usually via the mother's exposure to a substance.

First-order chemical reaction: Chemical reaction where the initial rate is directly proportional to the concentration of one of the reactants.

Gavage: Insertion of a tube into the esophagus to carry a substance directly to the stomach.

Genome: An organism's complete set of genetic material.

Genotoxic: Harmful to an organism's genetic material.

Ground spray: Applying a liquid using equipment designed for spraying from the ground.

Half-life: The time it takes for a chemical concentration to fall to one half of its initial value.

Hazard quotient: 1. Ratio of toxicant exposure (estimated or measured) to a reference value that is a toxicity threshold. 2. Ratio of estimated site-specific exposure to a single substance over a specified period to the estimated daily exposure level at which no adverse health effects are likely to occur.

Hazard ratio: A term used to compare the daily exposure to a chemical (e.g., pesticide) to its hazardous level, which has been adjusted by an uncertainty factor. A ratio greater than 1.0 indicates that the exposure is likely to have exceeded the benchmark safe level. Also see margin of exposure.

Hepatoxicity: Ability of a substance to harm the liver.

Herbicide: Substance intended to kill plants.

Human equivalent dose: Chemical dose believed to induce in humans the same magnitude of toxicity documented in an animal.

Immunological: Having to do with the immune system and innate/acquired immunity.

Immunotoxicant: A substance that can damage the immune system

Immunotoxicity: Adverse effects on the normal functioning of the immune system, caused by exposure to a toxic chemical.

Inert ingredient: An ingredient that was added intentionally but does not contribute to a mixture's *desired* biological effect; does not include impurities; may have *other* biological effects.

Latency: See latent period.

Latent period: Delay between exposure to a harmful substance and the manifestations of a disease or other adverse effects referable to the exposure.

Lipoprotein: Any of a group of conjugated proteins in which at least one of the components is a *lipid* (fatty acid or derivative of a fatty acid).

Margin of exposure: Ratio of the no observed adverse effect level (NOAEL) to the theoretical or *estimated exposure dose* or *concentration*.

Mutagenicity: Ability of a physical, chemical, or biological agent to cause heritable changes (mutations) in the genetic material (DNA) in a cell by changing or causing the loss of genes or chromosomes (or parts thereof).

Neurological: Having to do with the nervous system.

Neurotoxic: Capacity to produce adverse effects on the nervous system.

Overspray: Applying pesticide outside the target area.

Parts per trillion: For the purposes of this report, parts per trillion means $1/1,000,000,000,000 = 1 \times 10^{-12}$

Pesticide: A substance intended to kill pests, such as insects, weeds, and fungal pathogens; commonly, any substance used to control, prevent, or destroy animal, microbial, or plant pests.

Pharmacokinetics: The process by which the body takes in, biotransforms, distributes, and eliminates chemicals and their metabolites.

Pluripotent: 1. Able to affect more than one organ or tissue. 2. Able to develop in one of several ways.

Receptor: 1. Molecular structure in or on a cell that recognizes and binds to a compound and acts as a physiological signal or mediator of an effect. 2. A population, community, or ecosystem that is exposed to a contaminant or other stressor.

Receptor group: The at-risk population.

Reference dose (RfD): An estimate of the daily exposure of people, animals, or plants likely to be without risk of harmful effects, even if the exposure continues over a lifetime; these are: a) adjusted for sensitive sub-groups of the population, b) typically expressed in mg/kg bodyweight/day or µg/kg bodyweight/day, c) can be derived from a NOAEL, low observed adverse effect level (LOAEL), or benchmark dose, with uncertainty factors generally applied to reflect data limitations, d) generally used in U.S. EPA's non-cancer health assessments, and e) often used interchangeably with *acceptable daily intake*.

Referent (reference) population: The standard against which the population under study can be compared.

Registered pesticide: Pesticide products that the Pest Management Regulatory Agency has registered for use in Canada, with specific uses and conditions of use specified on the product label.

Relative absorption factor (RAF): The relative absorption difference between two exposure routes (e.g., oral and dermal), used to adjust exposure estimates for route-to-route extrapolation (e.g., converting an oral exposure limit to dermal one).

Relative risk (RR): risk ratio, rate ratio; 1. Ratio of the risk of disease or death among the exposed to the risk among the unexposed. 2. Ratio of the cumulative incidence rate in the exposed to the cumulative incidence rate in the unexposed.

Residue: Presence of a substance in an organism or other material such as food or packaging following exposure; may be deliberately added, as in herbicide residue on plants, or inadvertently added, as in dioxin residue in soil.

Risk assessment: The process of estimating the likelihood or chance that people or the environment will experience adverse effects from a series of events or circumstances, such as exposure to chemicals, substances, or agents.

Single nucleotide polymorphism: The most common type of genetic variation among people. Each represents a difference in a single DNA nucleotide.

Standardized mortality ratio (SMR): A ratio between the observed number of deaths in a study population and the number of deaths that would be expected based on the age- and sex-specific rates in a standard population and the age and sex distribution of the study population.

Teratogenicity: Ability to cause birth defects.

Threshold limit value: Concentration of a potentially toxic substance below which it is believed that a worker can be exposed during a work day over a working lifetime without adverse effect.

Tolerable daily intake: Estimate of the amount of a potentially harmful substance (e.g., contaminant) in food or drinking water that can be ingested daily over a lifetime without appreciable health risk. For regulation of substances that cannot be easily avoided, a *provisionally tolerable weekly intake (PTWI)* may be applied as a temporary limit.

Total toxic equivalent (TEQ): A TEQ is calculated by multiplying the actual grams weight of each dioxin and dioxin-like compound by its corresponding TEF (e.g., 10 grams X 0.1 TEF = 1 gram TEQ) and then summing the results. The number that results from this calculation is referred to as grams TEQ.

Toxic equivalency factor (TEF): The TEF is the ratio of the toxicity of one of the compounds in this category to the toxicity of the two most toxic compounds in the category, which are each assigned a TEF of 1: 2,3,7,8-tetrachlorodibenzo-p-dioxin (commonly referred to as dioxin) and 1,2,3,7,8-pentachlorodibenzo-p-dioxin. TEFs that have been established through international agreements currently range from 1 to 0.0001.

Toxicant: A toxic substance that is made or introduced into the environment by humans and harms organisms through physical/chemical interactions.

Toxicity: Ability to harm an organism, defined by the quantity administered or absorbed.

Toxicological reference value: An estimate of the dose associated with a specific level of risk or considered to be safe (in the panel's report, is usually a daily dose over a long period); is used to evaluate whether estimated/measured exposures are likely to cause adverse health effects and to develop guidelines and standards, such as drinking water quality guidelines.

Volatility: The readiness with which an element or compound will vapourize.

1. Executive summary

Beginning in the late 1940s to the late 1970s, the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) was used widely for brush control in Ontario, across Canada, and throughout the world. In Ontario, the herbicide was applied by or on behalf of provincial government departments and agencies, municipalities, and private citizens. The U.S. military used 2,4,5-T as a component of herbicide mixtures designed to defoliate forests during the Vietnam War. The most widely recognized of these herbicide mixtures was Agent Orange, a 50:50 mixture of 2,4,5-T and 2,4-D, so-called because of the identifying orange stripe on the outside of the drum.

The herbicide 2,4,5-T was contaminated with several chemical entities, the most problematic being 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), more commonly known as dioxin. By the early 1970s, the Government of Canada had begun to express concern about the possible health effects of exposure to 2,4,5-T and, notably, its dioxin contaminant. In 1979, recognizing these emerging health concerns in Canada and internationally, and to better control the use of 2,4,5-T, the Government of Ontario instituted a permit requirement for using 2,4,5-T in the province. As the health controversy continued to build, the province chose not to issue any permits, thereby preventing further use of 2,4,5-T in Ontario.

1.1. Charge to the Ontario Independent Fact-Finding Panel on Herbicide 2,4,5-T

On March 11, 2011, the Minister of Natural Resources announced the formation of the Independent Fact-Finding Panel on Herbicide 2,4,5-T. The panel was given the following mandate by the Government of Ontario:

- Investigate and document the scope and scale of the use of 2,4,5-T herbicide in the province by Ontario government ministries and agencies, including those acting as agents or as contractors
- Determine the specific time period when 2,4,5-T herbicide was used in the province by Ontario government ministries and agencies
- Determine the geographic area where 2,4,5-T herbicide was used in the province by Ontario government ministries and agencies
- Examine whether exposure to 2,4,5-T herbicide in the affected areas may have potential health impacts
- Document the methods 2,4,5-T herbicide was deployed by employees of provincial ministries and agencies, and the interaction of those employees and the general public with 2,4,5-T herbicide application operations in affected areas
- Review the preparation, application, and storage of 2,4,5-T herbicide as well as provincial occupational health and safety, and laws, standards and workplace practices including the use of personal protection equipment and applicable training in place at that time
- Refer, where appropriate, to the Workplace Safety and Insurance Board any findings that could assist its work

1.2. The panel's approach to its mandate

Dr. Leonard Ritter, professor (now emeritus) of toxicology in the School of Environmental Sciences at the University of Guelph, was appointed as chair of the panel. The first task for the panel chair was to identify the skills and expertise that would be required to fulfill the mandate of the panel and to recruit the most highly qualified individuals for the task. The recruitment process included direct contact with experts and announcements in scholarly media. Those who wished to serve were invited to submit expressions of interest highlighting relevant education and experience.

Following a thorough search, the panel chair advised the Minister of Natural Resources that he had appointed the following members:

- Dr. Jack Bend, distinguished university professor in the departments of pathology, physiology and pharmacology, and pediatrics at the Schulich School of Medicine and Dentistry, University of Western Ontario
- Dr. Aaron Blair, scientist emeritus at the U.S. National Cancer Institute, U.S National Institutes of Health, an expert in epidemiology.
- Mr. Elliot A. Sigal, president and senior scientist at Intrinsik Environmental Sciences, Inc., an expert in exposure assessment and risk assessment
- Dr. Jeanne Mager Stellman, professor emerita and special lecturer at the Mailman School of Public Health, Columbia University, an expert in exposure assessment

Citing personal reasons, Dr. Bend withdrew from the panel early in 2012. Dr. Nancy Kerkvliet, professor in the Department of Environmental and Molecular Toxicology and deputy director of the Environmental Health Sciences Center at Oregon State University, was then brought in as a toxicology expert.

The panel met several times in closed session to assign responsibilities within the panel, to review progress, and to discuss issues of relevance to its mandate. The panel also conferred frequently by email and phone, as required.

To assist the panel in its activities, a support office was established, staffed by two Ontario Public Service employees seconded from their home positions for the duration of the project. Staff in the support office reported directly and exclusively to the panel chair on all matters related to the panel. They provided administrative support to the panel and gathered additional information as requested by the panel members and the chair. The panel, with the assistance of the support office, also requested and received many other reports directly from various government departments.

1.3. Information reviewed by the panel

To ensure the panel could fulfill its mandate, all provincial ministries, the Cabinet Office, the Premier's Office and all boards, agencies, and commissions of the Government of Ontario were instructed (subject to any privilege or other legal restrictions) to help the panel to the fullest extent possible. They were told that the panel could request any information necessary to fulfill its mandate and that (subject to any privilege or other legal restrictions), government ministries should cooperate with and assist the panel.

In addition to the support office, a single coordinating office was set up in the Ministry of Natural Resources (MNR[1]) to ensure that government departments were consistent in responding and in collecting, scanning, and storing information. All government departments and agencies submitted information to the MNR coordinating office, where staff scanned records and organized them into a searchable database. Approximately 4700 records were submitted to the panel, in batches as they became available, with the final submission in September 2012. The panel also relied on the expertise of the individual panelists to identify important individual research publications, reports of internationally recognized expert agencies, and reports published by various government departments at the provincial, federal, and international levels to augment the nearly 4700 records it received from Ontario government departments and agencies. All records on which the panel relied are available to the public, on request, and all resource material used by the panel is cited in the bibliography of this report.

[1]In most cases, the names of organizations referred to in this report have changed several times during the more than three decades covering the period of use of 2,4,5-T in Ontario. The panel chose to refer to them by their most common or current designation.

1.4. Review of the toxicological and epidemiological evidence linking exposure to 2,4,5-T and TCDD to adverse health effects

Studies of the possible health effects from exposure to 2,4,5-T and TCDD have been reviewed by a number of respected national and international institutes and agencies. In addition, individual scientists have conducted independent reviews. Although the panel reviewed papers published by individual scientists, conclusions about potential health hazards from 2,4,5-T and TCDD were based primarily on the reviews and assessments of national and international organizations, including the Institute of Medicine (IOM), the United Nations World Health Organization, the U.S. Environmental Protection Agency (U.S. EPA), Health Canada, and the Ontario Ministry of the Environment (MOE). The panel was satisfied with the completeness and integrity of the institutional reviews and did not consider it necessary to re-assemble and re-evaluate the core studies on which the institutional reviews were based.

Similarly, the panel considered reports and reviews of experimental studies in laboratory animals that addressed the hazards of 2,4,5-T and TCDD in its overall assessment of potential adverse health effects. The panel did not attempt to derive its own tolerable daily intake values for TCDD or 2,4,5-T but was confident that the process and basis on which others had derived tolerable daily intake values for these compounds was sound.

As discussed in Chapter 6 of this report, the panel adopted a *threshold* model to characterize the potential for 2,4,5-T and TCDD to cause adverse health effects in humans. The threshold model is the internationally accepted basis for establishing safe exposure or dose levels for prescription drugs, residues in food, and contaminants in air and water. The approach is based on the presumption that a threshold exposure level can be identified below which adverse effects are not anticipated, even with lifetime daily exposure. The panel concluded that due to the lack of carcinogenicity associated with 2,4,5-T, the indirect carcinogenic effects of TCDD, and the general lack of mutagenicity associated with 2,4,5-T and TCDD, the threshold model was appropriate for application to 2,4,5-T and its contaminants.

The toxicity of 2,4,5-T and TCDD has attracted the attention of the scientific community for decades, and many publications are available on this topic. International consensus from both laboratory studies in animals and population studies in humans is that TCDD is a potent toxicant and potential human carcinogen, while exposure to 2,4,5-T, by itself, is associated with only limited toxicity. The United Nations Joint FAO/WHO Expert Committee on Food Additives has determined "...that a tolerable intake could be established for TCDD on the basis of the assumption that there is a threshold for all effects, including cancer...."

This determination is based on the indirect mechanism of action by which TCDD promotes cancer via activation of the *aryl hydrocarbon (Ah) receptor*. As described in Chapter 3, activation of the Ah receptor is considered an essential primary event that underlies all of the toxic effects associated with exposure to TCDD in both humans and laboratory animals. The United Nations Expert Committee also concluded that the establishment of a tolerable intake (a "safe" level) for TCDD based on effects other than cancer would also be protective of any cancer risk. The committee established a tolerable monthly intake of 74 pg/kg body weight (BW)/month, equivalent to a tolerable daily intake of 2.3 pg/kg BW/day. Health Canada and MOE have adopted the same tolerable intake of 2.3 pg/kg BW/day. For its risk assessment, the panel adopted the same tolerable daily intake level for TCDD as accepted by the governments of Canada and Ontario and the Joint FAO/WHO Expert Committee on Food Additives.

The panel also considered the need to establish a tolerable daily intake for the parent herbicide, 2,4,5-T. Since 2,4,5-T has little chronic toxicity, the panel adopted an acute tolerable daily intake of 20 µg/kg BW/day for short-term exposures to 2,4,5-T.

1.4.1. Exposure assessment

The panel used a two-part process to carry out the exposure assessment. The first step was an extensive review of every document in the MNR database that referred specifically to herbicide use. Information on where the chemical was used, how it was handled, and who was involved in the operations was extracted and summarized. Historical summary tables of spraying activities were constructed to estimate the volumes and concentrations used. Maps of the spraying were plotted to give a visual representation of the areas covered by historical spray activities. Exposure scenarios were developed for MNR spray programs to promote the regeneration of tree species with commercial value in northern Ontario; for Ontario Hydro maintenance of rights-of-way clearance around power transmission lines; and for Ministry of Transportation (MTO) maintenance to control weeds and brush along highways.

The exposure scenarios were then used to model potential human exposures to the chemicals of concern. Exposure assessment models took into consideration the chemical characteristics of 2,4,5-T and TCDD that determine how the chemical reacts in the ecosystem (e.g., how long does it persist? will it decompose in sunlight? what is the route of entry into the body?). Scenarios were created for the extent of impact of exposure from consumption of wild berries, plants, and wildlife; for aerial drift of sprayed herbicides; and for persistence of the chemicals in the soils and sediment in bodies of water. Because data on usage were incomplete, the panel used low, central, and high input parameter scenarios in its calculations to provide estimates of exposure across a range.

Exposure assessment modelling also took into account human physiological factors, such as body weight and breathing rate, which influence the extent to which a toxic substance may gain entry into the body and how it will interact once it enters. Where possible, the panel relied on standard values for environmental and physiological factors that were consistent with Health Canada and MOE. For occupational exposures, the panel used the U.S. EPA Occupational Pesticide Handler Unit Exposure Surrogate Reference Table and Pesticide Handler Exposure Database in its modelling. The occupational models take into account the use of protective equipment, and such equipment was part of MNR, Ontario Hydro, and MTO operating procedures. However, the panel had no way of knowing whether or not appropriate protective equipment was actually used, so again low, central, and high input parameter estimates were used.

The re-creation of exposure events that occurred decades ago is rife with unknowns and uncertainties. As such, many decisions were made along the way that influenced the outcome of the assessment. The quantitative—or numerical—exposure assessment required the input of many assumptions that affected large amounts of data and numerical variables. Some of these input variables were obtained from the published literature, while other information had to be scenario-specific and was obtained from the various sources of information available to the panel. Each of the decisions and input variables contain some element of variability and uncertainty, which can affect the final results and conclusions. The goal of quantitative exposure assessment was to produce a conservative model so that estimates of disease risks were not underestimated. Given the tendency for the assumptions used to overestimate exposure, it is likely that this assessment errs on the side of conservatism (safety).

1.4.2. Exposure from proximity to spraying

The likelihood and extent of exposure to the chemicals is a function of how close to spray activities an individual was. Herbicide spraying occurred in remote areas as well as close to more populated, residential areas. Remote spraying was generally by aerial application, while residential spraying was ground based.

MNR used herbicides primarily on forests in remote areas, with most spraying done aerially. MTO used ground-based methods to apply herbicides adjacent to roadways in both urban and rural areas. For the MTO scenario, properties were assumed to be located no closer than 30 m from the roadway. Ontario Hydro applied along

transmission corridors in both remote and populated areas via both aerial and ground-based methods. For the Ontario Hydro scenario, residential properties were assumed to be located no closer than 30 m from the right of way and recreational areas set back several hundred metres from the right of way.

The drift of herbicides from aerial spray was estimated using a standard model (AgDrift) and was used to estimate the concentrations in soil, food crops and wild berries, and wildlife contamination.

1.4.3. At-risk populations

The at-risk population (called *receptor groups* in the analysis) consisted of groups of people with potential exposures from occupational sources, from residences near sprayed areas or from recreational activities in or near sprayed areas (see list of receptor categories below). Given the many years over which herbicide spraying occurred, it is likely that many different individuals were occupationally involved in these activities for various periods between 1948 and 1979.

Occupational

- Mixer/loaders
- Applicators
- Flagmen
- Junior rangers
- Supervisors

Non-occupational

- Residents
- Recreational visitors
- Hunters/anglers
- First Nations people (dietary)
- Bystanders of aerial spray drift or overspray
- Spouses/other family members

Several possible exposure routes with the chemicals or with contaminated soil were considered in the assessment models. These included inhalation; ingestion of soil or contaminated game, fish, plants, or berries; and direct skin contact. Exposure to contaminated clothing was possible for several receptor groups, but because the panel thought it to be less likely and less easily quantifiable it was not included in the mathematical modelling.

Documentation of transport, storage, and disposal of the herbicides was also examined and summarized.

1.5. The development of standards and regulations for pesticide use in Ontario

As part of its mandate, the panel was asked to review the preparation, application, and storage of 2,4,5-T herbicide as well as provincial occupational health and safety laws, standards, and workplace practices, including the use of personal protective equipment and applicable training in place at that time.

Phenoxy herbicides were first used in North America in the late 1940s, and uses were largely unregulated. The first regulations focused on minimizing drift and damage to neighbouring crops rather than protecting the health and safety of applicators and bystanders. Standards and regulations were evolving from the 1950s, and language specific to protecting human health began to be considered in the 1970s, either via controlling occupational exposures or minimizing contamination of food intended for human consumption. Although the panel assessed and documented the evolution of pesticide use standards and regulations in Ontario, it could not determine the extent to which practices intended to protect the general public or to protect workers involved in pesticide handling, mixing, loading, and application were practiced, monitored, or enforced.

As early as 1954, Ontario Hydro produced the Manual of Spray Practices, which noted that "herbicides are known to be non-toxic to man and animals but operators should minimize breathing in the mist, or having it contact their faces." By 1962, the Ontario Hydro Manual of Spray Practices (A0141556[2]) was expanded to include matters of personal safety. In a 1965 circular, MTO listed neoprene gloves and blue coveralls as safety clothing available for weed spray crews. In 1976, the Ontario Ministry of Labour indicated in a memo that safety glasses and shields should always be worn; protective, impervious clothing was needed; and respiratory organic vapour canisters were required. The memo also noted that " ...workmen have been observed spraying with no protective clothing or equipment."

General concern about potential human health effects that might be associated with exposure to 2,4,5-T and its dioxin contaminant began to emerge in the early 1960s. In 1979, responding to safety concerns related to 2,4,5-T and the dioxin contaminant, the Government of Ontario instituted a permit requirement for using 2,4,5-T in the province. As the health controversy continued to build, the provincial government chose not to issue any permits, thereby preventing further use of 2,4,5-T in Ontario, six years prior to the de-registration of the herbicide by the federal statutory authority.

1.6. Conclusions of the panel

Exposure assessment is a key component of the risk assessment paradigm and was a critical aspect of the panel's undertaking. The use of 2,4,5-T in Ontario spanned a period of approximately 30 years. The panel carefully considered the issue of exposure and developed an exposure assessment effort to re-create an exposure profile for provincial workers and residents that could be used in an exposure-response evaluation, a basic premise of the toxicological risk assessment paradigm.

The panel adopted a well-documented and widely practiced risk assessment protocol to assess the potential health hazards of 2,4,5-T and TCDD, to evaluate the potential for exposure in various Ontario populations that could have arisen as a result of use by Ontario government departments and agencies, and to relate these exposure levels to exposure conditions under which adverse effects have been observed in studies of laboratory animals and from human populations. The panel could not locate any specific exposure monitoring data for humans related to uses of the herbicide by Ontario government departments and agencies. Lacking such direct information on exposure from herbicide applications by the provincial government, the panel carried out a comprehensive assessment of use records submitted by various departments and agencies to determine the locations and amounts of 2,4,5-T applied and to use this information to estimate likely human exposure to the herbicide and its contaminant during the period of use by the Ontario government. These application records indicated that while many departments used the herbicide to some extent, most uses by Ontario government departments and agencies were carried out by MNR, MTO, and Ontario Hydro. The panel assessment, therefore, focused on the uses by these three departments. The panel notes that most use of the herbicide in Ontario was not by the Government of Ontario but rather by private and municipal users. The assessment of these non-government uses was beyond the panel's scope and not included in the evaluations.

Because of the complexity of the exposure estimation process and the uncertainties associated with it, the panel provided a range of exposure estimates (a *low*, *central*, and *high* estimate) for each exposure scenario. These levels were based on assumptions included about each input parameter in the exposure assessment algorithm. The high-case estimate employed a series of individual worst-case assumptions for each component of the algorithm, applied one after another, introducing a repetitive input parameter selection bias. The high-case estimates

[2] Citations in the format *A0* refer to records that are contained within the MNR searchable database. These records are available to the public, subject to Canadian copyright provisions and the Freedom of Information and Protection of Privacy Act. To access these records, visit www.ontario.ca/245T.

represented the likely worst-case exposure situation. The low case estimates always took the low-end exposure parameters to generate the lowest likely overall exposure based on the information at hand. The central exposure estimate used intermediate (central or average) parameters to create a mid-level overall exposure estimate. Parameters used to develop exposure estimates included the amount of the dioxin contaminant in 2,4,5-T, the use of personal protective equipment, the fraction of applied herbicide penetrating the forest canopy, and the half-lives of TCDD and 2,4,5-T in soil. A range of potential exposure values (facilitated through the use of selected low, central, and high input parameters) were developed to provide a general appreciation for the level of uncertainty and variability present within the quantitative exposure estimates. Overall, the panel was of the view that the central estimate likely represented the most likely estimate of exposure.

The panel adopted a *margin of safety* approach to characterize the risk to various population subsets that may have been affected by the use of 2,4,5-T by Ontario government departments and agencies. With this approach, exposure levels of 2,4,5-T and TCDD that did not result in adverse health effects were identified from laboratory animal and human population studies. These exposure levels were adjusted by applying an uncertainty factor intended to compensate for the inherent uncertainty in the approach (for example, when studies in laboratory animals were used to predict effects in humans and inherent exposure assessment uncertainties in population studies in humans). This adjusted dose level was then directly compared with the level of estimated exposure the panel developed for different scenarios for various Ontario populations. Generally speaking, when this ratio was greater than 1, exposures may have exceeded a safe threshold; if less than 1, it was within the range of anticipated "no hazardous effect" based on the scientific literature.

The panel found situations where the benchmark reference exposure was exceeded for certain subgroups of the Ontario population. These exceedances were almost entirely restricted to chronic occupational exposures to the mid and highest levels of TCDD and from the central and high estimates for Ontario Hydro, MNR, and MTO. Where exceedances were observed at mid exposures to TCDD, they were typically marginal.

Bystander exposures, defined by the panel as being of brief nature and not from personal direct use of the herbicide, were found to exceed the benchmark level only in the case of Ontario Hydro and MNR and only for the highest TCDD exposure scenario. Mid and low bystander exposures to TCDD and all MTO bystander exposures were not found to exceed the benchmark margin. None of the bystander exposures to 2,4,5-T for Ontario Hydro, MNR, and MTO exceeded the benchmark level.

The panel also constructed maps of areas treated with the herbicide in Ontario to better visualize where potential opportunities for human exposure may have existed. These maps were developed from geographic information from spraying records provided by the ministries. The maps show that most spraying was remote from residences, and when standard models were applied, the risks for both exposure and for health effects for most situations in these areas were found to be well within the acceptable benchmark margins described in the report.

Given the results of the exposure assessment and margin of safety comparisons carried out by the panel, it was the panel's opinion that:

- The central estimate of exposure was the best estimate.
- Low and high estimates provided a useful approximation of the range of exposure to be considered because of the uncertainty in the estimation process.
- MTO: Exposures exceeded the benchmark for a relatively small number of individuals involved in ground mixing/loading and application by MTO employees.

- Ontario Hydro: Exposures exceeded the benchmark for some individuals involved in ground mixing/loading and application by Ontario Hydro employees.
- MNR: Exposures exceeded the benchmark for some individuals involved in backpack mixing/loading and application by MNR employees and junior rangers.
- MNR: Exposures exceeded the benchmark for some individuals involved in aerial mixing/loading and flagging by MNR employees.

Some bystander exposures exceeded the benchmark margin of 1, but the highest estimated exposures corresponded to margins of safety of less than 2. Given the assumptions and uncertainties in the bystander estimates, the acute nature of bystander exposures, and the safety factors inherent in the *toxicological reference values* (TRVs) used in the evaluation, the panel was of the view that the bystander margins of safety were not necessarily reflective of adverse health outcomes.

The panel considered an array of possible adverse health outcomes that have been reported to be associated with exposure to TCDD and/or 2,4,5-T. Although individuals exposed to these chemicals may have experienced some of these outcomes, it cannot be concluded that the outcome was due to the exposure. The risk assessment process used by the panel was of necessity directed at the population level and not intended to describe risks to any specific individual. The assessment revealed that several occupationally exposed populations may have experienced exposures above the margin of safety. However, such exposures were restricted to occupational populations (Ontario Hydro, MNR, and MTO workers) who were chronically exposed. As noted above, those whose exposures greatly exceeded the benchmark margin were almost all occupationally exposed populations who had the highest levels of TCDD exposure. The TRVs used to assess exposure acceptability erred on the side of safety, as regulatory policy for assessing risk requires, but populations with the highest exposure levels will not necessarily experience adverse health effects. The assessment merely indicated that acceptable margins of safety were exceeded, and people's health *could be* affected.

The panel's approach was a population level assessment and not intended to describe risks to any specific individual. A population level assessment provides the probability of occurrence of an adverse outcome in a population group but cannot deliver an absolute determination of occurrence of a disease in any individual. Individuals vary widely in their responses to exposures and in the exposures themselves. Having said this, the panel noted that the United States Institute of Medicine (IOM) has developed a list of diseases for which individuals exposed to 2,4,5-T and its contaminants may be at increased risk. The panel agreed that the IOM's findings reasonably reflect the current state of hazard assessment for these exposures. In Ontario, individuals with occupational exposures and a few bystander populations with the highest estimates exceeded the margin of safety benchmark. The associated risk of developing diseases as a result of 2,4,5-T or TCDD exposure for an individual in these categories would likely be very low. It is also important to note that an adverse effect will not necessarily occur, even in those cases where the margins the panel estimated have exceeded the benchmark.

2. Introduction

2.1. Background

Beginning in the late 1940s, the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) was used widely for brush control in Ontario, across Canada, and throughout the world. The U.S. military used 2,4,5-T widely as a component of herbicide mixtures designed to defoliate forests during the Vietnam War. By the early 1970s, the Government of Canada had begun to express concern about the possible health effects of exposure to 2,4,5-T and its contaminants (A0137447, Page 1; A0137444, Page 1; A0137443, Page 1).[1] In 1979, these health concerns led the Government of Ontario to impose a permit requirement for using 2,4,5-T in the province (O Reg 160/79, s 1). After that, no permits were issued in Ontario, preventing any further use of 2,4,5-T.

2.2. About the panel

On March 11, 2011, the Government of Ontario announced the formation of an independent fact-finding panel. The mandate of the panel was to:

- *Investigate and document the scope and scale of the use of 2,4,5-T herbicide in the province by Ontario government ministries and agencies, including those acting as agents or as contractors;*
- *Determine the specific time period when 2,4,5-T herbicide was used in the province by Ontario government ministries and agencies;*
- *Determine the geographic area where 2,4,5-T herbicide was used in the province by Ontario government ministries and agencies;*
- *Examine whether exposure to 2,4,5-T herbicide in the affected areas may have potential health impacts;*
- *Document the methods 2,4,5-T herbicide was deployed by employees of provincial ministries and agencies, and the interaction of those employees and the general public with 2,4,5-T herbicide application operations in affected areas;*
- *Review the preparation, application and storage of 2,4,5-T herbicide as well as provincial occupational health and safety, and laws, standards and workplace practices including the use of personal protection equipment and applicable training in place at that time; and*
- *Refer, where appropriate, to the Workplace Safety and Insurance Board any findings that could assist its work* (Province of Ontario 2011).

On March 11, 2011, then-Minister of Natural Resources Linda Jeffrey announced the appointment of Dr. Leonard Ritter, professor emeritus of toxicology in the School of Environmental Sciences at the University of Guelph, as panel chair. The chair's responsibilities included:

- establish the expertise needed for the panel to conduct its assessment
- develop a process to recruit suitably qualified members
- discuss the mandate of the panel with government authorities

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- establish timelines to complete the assessment and the scope of the review
- manage all aspects of panel function

The recruitment process included direct contact with experts and announcements in scholarly media (Appendix 2.1). Those who wished to serve were asked to submit expressions of interest highlighting relevant education and experience. Following a thorough search, the panel chair advised the Minister of Natural Resources that he had appointed the following members:

- Dr. Jack Bend, distinguished university professor in the departments of pathology, physiology and pharmacology, and pediatrics at the Schulich School of Medicine and Dentistry, University of Western Ontario
- Dr. Aaron Blair, scientist emeritus at the U.S. National Cancer Institute, U.S. National Institutes of Health, an expert in epidemiology
- Mr. Elliot A. Sigal, president and senior scientist at Intrinsik Environmental Sciences, Inc., an expert in exposure assessment and risk assessment
- Dr. Jeanne Mager Stellman, professor emerita and special lecturer at the Mailman School of Public Health, Columbia University, an expert in exposure assessment

Citing personal reasons, Dr. Bend withdrew from the panel early in 2012. Dr. Nancy Kerkvliet, professor in the Department of Environmental and Molecular Toxicology and deputy director of the Environmental Health Sciences Center at Oregon State University, was then brought in as a toxicology expert.

More information about the panel is available on the panel's website:

http://ontarioindependentfact-findingpanelon245t.ca/whoweare.php

2.3. How the panel acquired its information

To ensure the panel could fulfill its mandate, all ministries, the Cabinet Office, the Premier's Office, and all boards, agencies, and commissions of the Government of Ontario were instructed (subject to any privilege or other legal restrictions) to help the panel to the fullest extent possible. They were told that the panel could request any information necessary to fulfill its mandate and that (subject to any privilege or other legal restrictions) government ministries should cooperate with and assist the panel.

To ensure that government departments were consistent in responding and in collecting, scanning, and storing information, a single coordinating office was set up in the Ministry of Natural Resources (MNR). All government departments and agencies submitted information to the MNR coordinating office, where staff scanned records and organized them into a searchable database of approximately 4700 records. They delivered the database to the panel in phases; the final submission occurred in September 2012.

A support office was established to assist the panel in their activities. The staff in the support office consisted of two Ontario Public Service employees seconded from their home positions for the duration of the project. Staff in the support office reported directly and exclusively to the panel chair on all matters related to the panel, gathered additional information as requested by the panel members and chair, and provided administrative support to the panel. The panel, with the assistance of the support office, also requested and received many other reports directly from various government departments.

To meet the panel's mandate, panelists were assigned responsibilities for various sections of the report and worked independently on them. A secure electronic portal was established for communication by email and telephone and for document exchange. The panel also held several face-to-face meetings to develop work procedures, evaluate data, and prepare reports in order to answer the questions posed by the Government of Ontario.

The panel originally expected to release its report and findings to the general public and the Minister of Natural Resources in summer 2012. However, due to the time required to search and assess thousands of records spanning more than three decades, the panel could not meet this target date, and the release was delayed. Upon evaluating the completeness of the records, the panel members were satisfied that they had the full cooperation of all Government of Ontario departments and agencies for the following reasons:

(1) Massive amounts of information were made available.

(2) There was consistent information between contemporary summary information and individual spray reports.

(3) When requested by the panel, the government exerted its best efforts to obtain additional records.

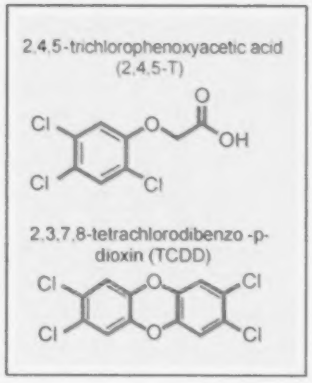
2.4. Assessment scale

The panel's goal was to assess potential exposure and any possible associated risks that provincial government use of the herbicide 2,4,5-T poses to population health. The approach taken by the panel was directed at the population level and is not intended to be interpreted at the level of an individual. However, the panel is of the view that the range of exposure captured at the population level likely included the range of individual exposures. The panel had indicated early in its deliberations that they would not accept or respond to submissions from individuals. The panel did not have the legal structure to protect personal information and cautioned that personal information (such as medical records) should not be submitted. All submissions to the panel from individuals or organizations were redacted by the panel support office before submission to the panel in order to protect privacy.

2.5. Assessment scope

2.5.1. Chemical scope

Like all pesticides, commercial herbicides are typically made up of an active ingredient along with other chemicals such as solvents, emulsifiers, and at times, inadvertent contaminants. For 2,4,5-T specifically, polychlorinated dibenzo-*p*-dioxins can form under poorly controlled manufacturing conditions, with the formation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) of greatest concern. Much of the worldwide debate on the health effects of 2,4,5-T has centred on the toxicity associated with its TCDD contamination.



Herbicides have historically been widely used in Ontario and around the world, in agricultural settings, industrial and commercial applications, forestry, and urban landscapes (urban pesticide use was banned in Ontario in 2009). Depending on the vegetation to be controlled, and on the specific setting, many different herbicides, alone or in various combinations, may be applied. 2,4,5-T, a synthetic plant auxin, was widely used in Canada and throughout the world over approximately three decades.

When used in Ontario, 2,4,5-T was often combined with other herbicides, notably 2,4-dichlorophenoxyacetic acid (2,4-D). However, the issue of TCDD contamination and related health effects was restricted to 2,4,5-T. The panel mandate was 2,4,5-T. Other widely used herbicides, such as 2,4-D and 2,4,5-TP, were not included in this assessment. Similarly, 2-(2,4,5-trichlorophenoxy) propionic acid (2,4,5-TP), a different herbicide from 2,4,5-T and sometimes also known as fenoprop or silvex, was not included in the assessment.

As for Agent Orange, this term is often used interchangeably with 2,4,5-T and has been used by others to refer to the panel's work. Agent Orange was a combination of herbicides—a 50/50 mix of 2,4,5-T and 2,4-D—that the U.S. military used during the Vietnam War. The U.S. military coined the term *Agent Orange* based on the orange stripe used to identify the containers. The combination of 2,4,5-T and 2,4-D was widely used in Ontario.

As for Agent Orange, this term is often used interchangeably with 2,4,5-T and has been used to refer to the panel's work. Agent Orange was a combination of herbicides—a 50/50 mix of 2,4,5-T and 2,4-D—that the U.S. military used during the Vietnam War. The combination of 2,4,5-T and 2,4-D was widely used in Ontario.

2.5.2. User scope

The panel mandate included use of 2,4,5-T by Ontario government departments and agencies but did not extend to private users or municipalities, which applied most of the 2,4,5-T in Ontario. In 1978, the Ontario Pesticide Advisory Committee reported that Ontario municipalities used almost 90,000 kg of a 50:50 2,4-D + 2,4,5-T mixture (about 45,000 kgs of 2,4,5-T). The provincial ministries currently known as Ministry of Transportation (MTO) and MNR, and the organization commonly known as Ontario Hydro[1] combined used approximately 23,000 kgs of 2,4,5-T, about half the amount municipalities used that year (Figure 2.1). Although the panel was unable to determine if these 1978 data were representative of uses in other years, they had no indication to the contrary. The panel did not identify manufacturing or ordering procedures that were specific to Ontario government operations.

The panel mandate included use of 2,4,5-T by Ontario government departments and agencies but did not extend to private users or municipalities, which applied most of the 2,4,5-T in Ontario.

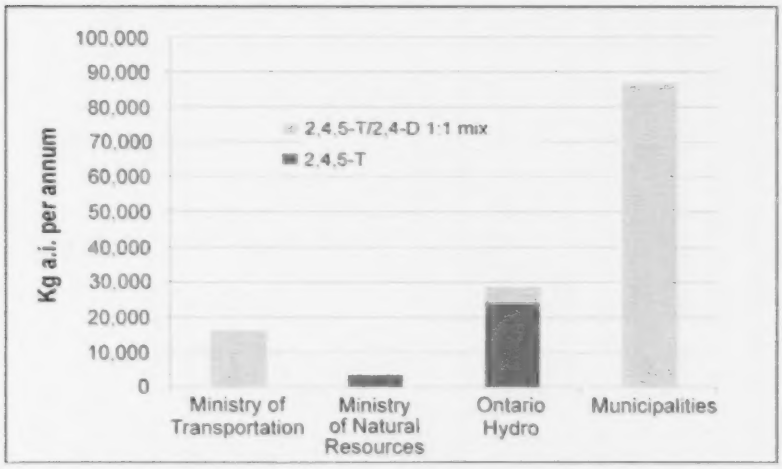


Figure 2.1. *Annual[a] use of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) by provincial government agencies, as reported in the 1978 Ontario Pesticide Advisory Committee report Review of the Use of 2,4-D, Other Phenoxy Herbicides and Picloram by Ontario Government Agencies (A0133096).*

[a]The panel was unable to determine what year annual referred to and how representative these use patterns were throughout the period of use of 2,4,5-T in Ontario.

[1]In most cases, the names of organizations referred to in this report have changed several times during the more than three decades covering the period of use of 2,4,5-T in Ontario. The panel chose to refer to them by their most common or current designation.

The panel did not find comprehensive records that specified suppliers of 2,4,5-T for government departments or agencies, but the Ontario government and its agencies could likely have bought 2,4,5-T from several sources. The panel was satisfied that the 2,4,5-T purchased by Ontario government departments and agencies would have been similar, with regard to its contaminant levels, to the herbicide purchased by the U.S. military and other industrial and commercial users. The panel did not identify manufacturing or ordering procedures that were specific to Ontario government operations.

The panel was satisfied that the 2,4,5-T purchased by Ontario government departments and agencies would have been similar, with regard to its contaminant levels, to the herbicide purchased by the U.S. military and other industrial and commercial users.

2.6. Assessment report structure

This report is divided into several sections, which address the questions raised by the Government of Ontario for the panel to consider. The issues the panel assessed included:

- where 2,4,5-T was used in Ontario
- how and how much was used
- when and by whom it was used
- the toxicology of 2,4,5-T and TCDD
- the epidemiological evidence linking human exposure to 2,4,5-T and TCDD with adverse health outcomes
- exposure in various populations who may have applied or been otherwise exposed to 2,4,5-T and its TCDD contaminant
- how standards, policies, practices, guidelines, and regulations about the use of pesticides in Ontario evolved

While the panel carefully assessed and summarized the evolution of standards, policies, practices, guidelines, and regulations in Ontario regarding the use of pesticides, the members could not determine the extent to which they were followed or enforced.

2.7. About risk assessment

Risk assessments are conducted to understand the relationship between exposure and disease. *Risk* is the expression of the inherent toxicological properties of the putative agent (*hazard*) as a function of the exposure to this agent. The panel adopted the risk assessment approach that the U.S. National Academy of Sciences (NAS) first advanced in 1983 (NRC 1983), as it appeared to be well suited to its mission. This approach addresses four key elements of assessing human health risks:

- **Identifying hazard:** Describing the inherent toxicological properties of the agent without regard for the exposure circumstances under which the toxicological endpoints (hazards) are expressed
- **Assessing dose response:** Describing what effects the chemical might have across a wide range of doses or exposures, which is critical to determining if the toxicological endpoints observed during hazard identification are likely to occur during practical use of the chemical

- **Assessing exposure:** Describing exposure during typical use of the chemical, to inform a biologically meaningful assessment of whether the exposure is of a magnitude, frequency, and duration that might reasonably be expected to be associated with an adverse health effect
- **Characterizing risk:** Integrating hazard identification, dose response assessment, and exposure assessment to predict and describe the likelihood of an adverse health outcome

To understand a dose response relationship, exposure must be characterized over many years. This requirement is challenging in epidemiologic studies because actual internal dose or environmental measurements are often not available. Thus, human exposures must be reconstructed using limited measurements supplemented with exposure determinants, and often for past decades.

The panel faced these difficulties with 2,4,5-T exposure; few exposure measurements were available, and the most recent uses of 2,4,5-T in Ontario occurred more than 30 years ago. Thus, the panel had to reconstruct several exposure scenarios (their approach is detailed in other sections of this report). The panel chose to describe possible exposure scenarios arising from herbicide use by Ontario Hydro, MTO, and MNR. While several other departments and agencies were involved in applying 2,4,5-T, the uses by these other departments and agencies were considered by the panel to be very minor. The panel therefore concluded that use by Ontario Hydro, MTO, and MNR represented most of Ontario government departments'/agencies' use of 2,4,5-T.

The panel chose to describe possible exposure scenarios arising from herbicide use by Ontario Hydro, MTO, and MNR. These users represented most of Ontario government departments'/agencies' use of 2,4,5-T.

To estimate historical occupational and bystander exposures without empirical data, the panel relied on several models, which are detailed elsewhere in this report:

- Pesticide Handler Exposure Database (US EPA 2012)
- Government of Australia (2010)
- U.S. Forest Service of the U.S. Department of Agriculture (Spray Drift Task Force 2000)

2.8. About thresholds

A key scientific question related to understanding the biology of chemically induced cancers is whether a *threshold* or *no-effect level* exists for some carcinogens. Traditionally, many researchers believed that any exposure to a carcinogen would result in some risk, although very small at very low exposure levels. Known as the *non-threshold* or *one hit* model, this hypothesis held that zero risk is possible only with zero exposure. Any exposure greater than null would result in some level of risk to exposed populations, especially for *genotoxic* carcinogens, which interact directly with and damage DNA. However, some scientists argue that this approach or hypothesis fails to account for repair mechanisms that can identify and remove damaged DNA that would otherwise turn into cancer/tumours.

The alternative is the *threshold response* approach, which holds that chemical exposure below a toxicologically relevant level (*threshold*) is not associated with adverse effects. Historically, the threshold approach has not been used to estimate risks from carcinogen exposure, but more recent advances in risk assessment science have led the scientific community to discuss integrating the threshold and non-threshold models for the risk assessment of carcinogens.

The strengths and weaknesses of toxicologic and epidemiologic studies are complementary; together they provide the best opportunity to understand exposure-disease relationships.

2.9. Scientific uncertainty of risk assessment

Identifying and managing scientific uncertainly in each step of risk assessment is challenging. A few examples of sources of uncertainty:

- the default parameters and values used to assess risks
- incomplete knowledge of effects and exposures
- inter- and intra-species extrapolations for dose responsiveness and biological susceptibility

The panel was aware of how scientific uncertainty could affect their assessment and conclusions. They have noted the potential impact of uncertainty in all relevant sections of this report.

The panel's approach was a population level assessment and not intended to describe risks to any specific individual. A population level assessment provides the probability of occurrence of an adverse outcome in a population group but cannot deliver an absolute determination of occurrence of a disease in any individual. Individuals vary widely in their responses to exposures and in the exposures themselves. Having said this, the panel notes that the United States Institute of Medicine (IOM) has developed a list of diseases for which individuals exposed to 2,4,5-T and its contaminants may be at increased risk. The panel agrees that the IOM findings reasonably reflect the current state of hazard assessment for these exposures. In Ontario, individuals with occupational exposures and a few bystander populations with the highest estimates exceeded the margin of safety benchmark. The associated risk of developing diseases as a result of 2,4,5-T or TCDD exposure for an individual in these categories would likely be very low. It is also important to note that an adverse effect will not necessarily occur, even in those cases where the margins estimated by the panel have exceeded the benchmark.

3. Review of the epidemiological and toxicological evidence related to potential health effects of 2,4,5-T and TCDD

3.1. Charge question

As part of its mandate, the Independent Fact-Finding Panel on Herbicide 2,4,5-T was asked to:

> *examine whether exposure to 2,4,5-T in the affected areas may have potential health impacts* (Province of Ontario 2011).

3.2. Introduction

To address this charge, the panel reviewed the epidemiological and toxicological literature to evaluate the evidence for possible health hazards from exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and associated contaminants. A full assessment of potential adverse human health effects relies on both experimental laboratory studies and observational epidemiological investigations because of their complementary strengths and weaknesses.

Experimental studies enable scientists to precisely characterize and control exposures and use a randomized study design to control for potential study biases. In addition, being able to deliver exposure precisely enables scientists to characterize exposure-response relationships in detail and to identify and clarify how toxicants act in living systems. However, findings from laboratory and animal experiments must be extrapolated to human populations, which can be challenging.

Epidemiological studies require no such extrapolation and provide direct evidence on disease risks from exposures to humans. On the other hand, characterizing exposure levels and controlling for potential biases is challenging in observational epidemiological studies.

When both experimental and epidemiological data point to an association, then scientists have the greatest confidence in the evaluation of the chemical's hazards to humans.

Epidemiological and toxicological studies of 2,4,5-T and its contaminant 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) have produced a wealth of information. Because epidemiological studies are observational, they always involve both the active ingredient 2,4,5-T as well as various contaminants such as TCDD and added formulants and inert ingredients. TCDD persists longer in the environment and in living organisms, including humans, so it is easier to perform exposure-response assessments for TCDD than for 2,4,5-T in epidemiological studies. In an epidemiological study, exposure-response gradients can be used to separate effects of 2,4,5-T and TCDD when the exposures are not correlated or are weakly correlated. However, this is unlikely for 2,4,5-T and TCDD. Thus, it is not possible to connect a particular health effect to any specific chemical exposure associated with use of 2,4,5-T alone.

In an epidemiological study, exposure-response gradients can be used to separate effects of 2,4,5-T and TCDD when the exposures are not correlated or are weakly correlated. However, this is unlikely for 2,4,5-T and TCDD. Thus, it is not possible to connect a particular health effect to any specific chemical exposure associated with use of 2,4,5-T alone.

Studies of the possible health effects of exposure to 2,4,5-T and TCDD have been reviewed by many national and international institutes and agencies (e.g., IARC 1977, 1987, 1997; WHO 1998; CCFME 2002; NRC 2006; NTP 2011; IOM 2012; US EPA 2012). Individual scientists have also performed reviews (Cole et al. 2003, Crump et al. 2003, Starr 2003, Steenland et al. 2004, Guernsey 2007, Boffetta et al. 2011). Although the panel assembled and reviewed papers by individuals and considered their conclusions, they relied primarily on the reviews and assessments of national and international organizations, for the following reasons:

- These organizations have documented procedures for their hazard assessments, which were conducted by international groups of distinguished scientists. Established procedures ensure the highest scientific rigour and integrity.
- Their reviews provide a measure of consistency that could be lacking in ad hoc evaluations conducted without *a priori* assessment guidelines.

Where appropriate, the panel used comments and conclusions from individual studies when they raised important scientific issues or provided data not available in the agency/institutional reviews.

3.3. Assessment of the potential for health hazards from exposure to 2,4,5-T and TCDD—organizations and agencies

The panel defines *assessment of health hazards* as an approach to determine the intrinsic ability of a substance to cause a human disease or ill health effect. The term *cause* refers to causal relationships that link exposures to the chemicals of concern with the health effects they produce, i.e., determining causality (Porta 2008). Causal inference is used to assess or test whether a relation of cause to effect does or does not exist. It implies that a change in the exposure level would result in some change in the effect level.

For 2,4,5-T and TCDD, several diseases, or outcomes, have been evaluated in the literature, including cancer, heart disease, neurological conditions, reproductive and immunological outcomes, endocrine function, and skin conditions. Although characterizing disease risk at a specific exposure level is not the goal of hazard assessment, information on exposure-response relationships from bioassays and epidemiological studies are considered when evaluating whether a hazard exists.

Table 3.1 lists the previous reviews of epidemiological and experimental studies that the panel used in its evaluation of whether 2,4,5-T and/or TCDD show a causal relationship to a harmful effect in humans and other species. Chapter 6 presents the panel's assessment of the possible disease risk from specific dose/exposure levels.

Table 3.1. *Human health effects from exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and dioxins, including 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Source	Chemical of concern	Health endpoint			
		Cancer	Heart disease	Neurological outcomes	Other
IARC 1977	2,4,5-T and esters	• no evaluation possible from animal/human data			
IARC 1997	2,3,7,8-TCDD	• carcinogenic to humans • excesses for non-Hodgkin lymphoma, lung cancer, all cancers combined			
IARC 1987	Chlorophenoxy herbicides	• limited evidence • cancers linked in epidemiological studies—soft tissue sarcoma, non-Hodgkin lymphoma, Hodgkin disease			
ACGIH 2001	2,4,5-T	• not classifiable as a human carcinogen			
WHO 2003	Chlorophenoxy herbicides in drinking water	• limited evidence—association with non-Hodgkin lymphoma (but not soft tissue sarcoma, Hodgkin disease) • provided guideline for levels in drinking water			• evidence for associations with miscarriages/birth defects in cross-sectional studies but not in cohort studies • provided guideline for levels in drinking water
NRC 2006	Dioxin and related compounds	• dioxin—committee split over classification as carcinogenic in humans or likely to be carcinogenic in humans, based mainly on total cancer • dioxins other than TCDD—committee agreed with classification of likely to be a human carcinogen	• not convincingly demonstrated	• available studies—no clear link between dioxin and risks of individual, clinically significant, non-cancer endpoints	• clear association with chloroacne; suggestive of Type II diabetes • prudent for the U.S. EPA to regard TCDD as immunotoxicant • inconclusive re: whether/in what way dioxin-like compounds immunotoxic in humans
IOM 2012	Agent Orange and dioxins	• sufficient evidence—soft-tissue sarcoma (including heart), non-Hodgkin lymphoma, chronic lymphocytic leukemia • limited evidence—multiple myeloma; cancer of larynx, lung, prostate	• limited evidence—ischemic heart disease, hypertension	• limited evidence—early-onset transient peripheral neuropathy, Parkinson disease	• sufficient evidence—chloracne, amyloid light-chain amyloidosis, porphyria cutanea tarda, Type 2 diabetes, spina bifida
NTP 2011	TCDD	• known human carcinogen • excesses of non-Hodgkin lymphoma, lung cancer, all cancers combined			

Source	Chemical of concern	Health endpoint			
		Cancer	Heart disease	Neurological outcomes	Other
US EPA SAB 2011	TCDD	• SAB[b] agreed with U.S. EPA[c] conclusion that TCDD is carcinogenic to humans • In developing oral slope factor (risk from lifetime of oral exposure) for TCDD using results of Cheng et al. (2006) study based on total cancer mortality • Ontario panel agreed with this approach			• SAB supports U.S. EPA selection of Mocarelli et al. (2008) and Baccarelli et al. (2008) studies for identifying co-critical effects for deriving reference dose
Cogliano et al. 2011	Dioxin and dibenzofuran	• 2,3,7,8 TCDD carcinogenic to humans for all cancers combined • limited evidence for soft tissue sarcoma, non-Hodgkin lymphoma, lung cancer • 2,3,4,7,8-pentachlorodibenzofuran—carcinogenic to humans			
Individuals					
Johnson 1992	TCDD	• no causal link with malignant lymphomas, soft-tissue sarcomas			
Starr 2003	Dioxin	• predicted zero excess cancer deaths from all dioxin exposures			
Cole et al. 2003	Dioxin	• TCDD not carcinogenic to humans at low levels			
Steenland et al. 2004	Dioxin	• epidemiological and toxicological data available since 1997 further supports conclusion as carcinogenic to humans • TCDD levels near general background may be carcinogenic			
Humblet et al. 2008	Dioxin		• associated with ischemic heart disease, total cardiovascular disease • stronger with ischemic heart disease		
Boffetta et al. 2011	TCDD	• animal experiments—carcinogenicity may be plausible • epidemiological data—far short of demonstrating relationship in humans			

[a] Blank cells = information not available. [b] SAB = Science Advisory Board. [c] U.S. EPA = U.S. Environmental Protection Agency.

3.3.1. International Agency for Research on Cancer (IARC)

The International Agency for Research on Cancer (IARC) has performed several reviews of 2,4,5-T and/or dioxins. IARC reviewers follow an established procedure for evaluating the potential of various substances to cause cancer in humans. The agency established one of the first procedures for systematically evaluating cancer risk from various exposures. A detailed description of IARC review purposes, processes, and procedures can be found in the preamble of each monograph and at the monograph website (IARC 2006). The process includes assembling scientific literature on:

- studies on humans and experimental animals
- mechanisms and other relevant toxicological data
- possible human exposure

This literature is evaluated by an IARC working group of 15 to 20 scientists selected using the following criteria:

- knowledge and experience in relevant disciplines
- lack of real or apparent conflict of interest
- geographic diversity
- balance of scientific views

Others who may support working group activities or attend working group meetings are:

- experts with critical knowledge of specific topics
- other observers with relevant scientific credentials
- IARC secretariat scientists
- representatives of national and international organizations

These other participants may present scientific information and discuss the evidence but do not participate in the evaluations. Before each meeting, the working group members prepare draft documents that summarize the literature. Then over the several days of the meeting, those attending discuss and modify the drafts and make an evaluation regarding carcinogenicity. By the time the meeting is adjourned, the working group has completed the final draft and conclusions.

The IARC conducted its first review of 2,4,5-T in 1977. The working group found only one study of workers with potential exposure and concluded that not enough evidence was available to make an evaluation (IARC 1977). IARC (1987) provided an evaluation of chlorophenoxy herbicides, a chemical group that includes 2,4,5-T as well as other phenoxy herbicides, but not of the carcinogenicity of specific phenoxy herbicides or their contaminants. This unspecified group of chlorophenoxy herbicides were assigned to Group 2B (*possibly carcinogenic to humans*) based on limited evidence for humans and inadequate evidence for 2,4,5-T in animals. Some cohort studies showed increases in soft tissue sarcoma and lung cancer, and some case-control studies showed soft tissue sarcomas and lymphomas, thus providing limited evidence for carcinogenicity from human studies.

In 1997, IARC concluded that TCDD was carcinogenic to humans (Group 1) based on evidence of multisite carcinogenicity in animals and limited evidence from human cohort studies of an excess of all cancers combined (IARC 1997). The working group noted that some cohorts had positive exposure-response trends with an increase in lung cancer in highly exposed subgroups. They also noted that some cohort and case-control studies of

herbicide applicators with relatively low exposures had excesses, but they did not consider these results crucial for the evaluation.

In a recent update on more than 100 Group 1 agents, IARC concluded there was:

- Sufficient evidence to conclude TCDD was associated with all cancers combined.
- Limited evidence to conclude that TCDD was associated with soft-tissue sarcoma, non-Hodgkin lymphoma, and lung cancer (Cogliano et al. 2011).

3.3.2. World Health Organization

The World Health Organization (WHO 2003) conducted a literature review related to cancer and reproductive effects from chlorophenoxy herbicide exposure in drinking water. Their conclusions were as follows:

- Animal and human studies provided some evidence for carcinogenicity and for reproductive effects from chlorophenoxy herbicide exposure.
- This literature could be used to set guidelines for levels in drinking water.

3.3.3. Institute of Medicine

The Institute of Medicine (IOM) of the National Academy of Sciences (NAS) serves as an adviser to the U.S. government on issues related to medical care, research, and education. In 1991, the U.S. Congress instructed IOM to periodically review how herbicide exposure could be affecting Vietnam veterans and whether a plausible biological mechanism or other evidence of a causal relationship exists (IOM 2012). IOM's first report was in 1994 (IOM 1994), with subsequent reports about every two years and the most recent in 2010 (IOM 2012). The most recent IOM committee to review the health effects of herbicide exposure in Vietnam veterans comprised 16 scientists who reviewed the epidemiological and toxicological literature on this topic through September 2010 and prepared a report on their conclusions. The draft report was reviewed by 15 other scientists. The committee determined the following[1]:

- Sufficient evidence was available to conclude that herbicides used in Vietnam (and their contaminants) were associated with development of soft-tissue sarcoma, non-Hodgkin lymphoma, chronic lymphocytic leukemia, Hodgkin disease, and chloroacne.
- Limited or suggestive evidence showed an association with larynx, lung, and prostate cancer; multiple myeloma; amyloidosis; early-onset peripheral neuropathy; Parkinson disease; porphyria cutanea tarda; hypertension; ischemic heart disease; and Type 2 diabetes (IOM 2012).
- Evidence suggested occurrence of spina bifida in children of exposed people.
- Limited or suggestive evidence was available for a classification of no evidence of an association between parental exposure to TCDD and *spontaneous abortion* (miscarriage before 20 weeks).
- Insufficient evidence was available to determine effects on infertility, spontaneous abortion, birth defects, Parkinson disease, endometriosis, neurobehavioural disorders, immune system disorders, leukemia, and cancers of the oral cavity, pharynx, nasal cavity, esophagus, stomach, colon, rectum, liver and biliary tract, pancreas, bone, breast, bladder, kidney, brain, and endocrine system (Appendix 3.1).

[1]The IOM uses *sufficient evidence* to refer to associations for which chance, bias, and confounding could be ruled out with reasonable confidence and *limited evidence* to refer to associations for which chance, bias, and confounding could not be ruled out.

3.3.4. U.S. National Toxicology Program Report on Carcinogens

The U.S. National Toxicology Program (NTP) publishes a biennial Report on Carcinogens to identify agents, substances, mixtures, or exposure circumstances that could pose a cancer hazard to humans (NTP 2011). Information from experimental toxicology and epidemiology studies is used in these evaluations. The reviewers use the following classifications:

- *Known to be human carcinogen*—used when sufficient evidence is available to show an exposure has a causal relationship with cancer—in other words, the association cannot be explained by chance, bias, or confounding.
- *Reasonably anticipated to be human carcinogen*—used when evidence shows that:
  - *A causal interpretation is credible but alternative explanations (chance, bias, or confounding factors) could not be excluded.*
  - *Sufficient evidence is available for multiple experimental animals, based on multiple exposure routes or an unusual degree of exposure by incidence, type of tumour, or age at onset.*
  - *The substance belongs to a class of chemicals known to be carcinogens but only some (less than sufficient) evidence is available for humans or laboratory animals.*

NTP staff follow the procedures below to evaluate substances for the Report on Carcinogens:

- review the scientific literature and develop a background document (often with the help of consultants with expertise/knowledge of the substance being evaluated)
- have an expert panel review the draft report at a public meeting
- have two independent federal committees conduct an internal review

The 2011 Report on Carcinogens lists TCDD as a *known human carcinogen* based on:

- sufficient evidence of carcinogenicity from epidemiological studies in humans that found increases in overall cancer mortality in four industrial cohorts
- a scientific consensus that a common mode of action for TCDD and other chlorinated dibenzodioxins and dibenzofurans involves the initial binding to the aryl hydrocarbon (Ah) receptor

3.3.5. U.S. Environmental Protection Agency (U.S. EPA)

The U.S. EPA evaluates potential health risks to humans from various environmental chemicals and presents these evaluations via the Integrated Risk Information System (IRIS). The IRIS process involves:

- developing a draft review of the scientific literature for the chemical of concern
- conducting internal and external scientific reviews of the draft document
- developing U.S. EPA responses to reviewer comments
- posting an IRIS summary and final toxicological review on the U.S. EPA's web site (US EPA 1994, 2012)

In 2006, the U.S. EPA asked the National Research Council (NRC; part of the NAS) to review the 2003 version of the U.S. EPA assessment of the health effects of TCDD and related compounds. NRC was to address:

- the scientific evidence for classifying TCDD as a human carcinogen
- the validity of the non-threshold linear dose-response model and the cancer slope factor the U.S. EPA calculated through use of this model

A non-threshold carcinogen is usually genotoxic and is presumed to have no safe dose. A threshold model is based on the assumption that developing cancer requires a minimum level of exposure (the *threshold*), below which no cancer will develop.

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The NRC committee conducting the review of the EPA evaluation comprised 18 scientists (NRC 2006), and the document they prepared was reviewed by another 15 scientists. NRC findings were as follows:

- They were split on whether to label TCDD as carcinogenic to humans or likely to be carcinogenic to humans, but they concluded that both descriptions had similar implications for public health.
- They concluded that other dioxins should be classified as likely to be carcinogenic to humans and agreed with the U.S. EPA that TCDD is likely to be a human immunotoxicant at "some dose level."
- They agreed with the U.S. EPA that TCDD probably causes reproductive and fetal development problems in rodents; however, they also indicated that more information is needed to show how animal findings are relevant to humans.
- They found that the U.S. EPA's decision to use only the non-threshold model for extrapolation was not adequately supported and recommended that U.S. EPA use both threshold and non-threshold models to extrapolate risks at lower levels.
- They also concluded the U.S. EPA did not adequately quantify the uncertainties associated with exposure assessment and response at a particular effective dose.

The U.S. EPA asked its Science Advisory Board (SAB) to review the agency's responses to the National Research Council's evaluation (NRC 2006) of the U.S. EPA dioxin document. The SAB (US EPA SAB 2011) found that:

- U.S. EPA's considerations of the epidemiological and animal bioassay data were scientifically justified and applied in a scientifically sound manner.
- U.S. EPA's characterization of TCDD as carcinogenic to humans was correct, although one board member dissented.
- It was appropriate to use a physiologically based pharmacokinetic model to evaluate the internal dose of TCDD in human and rodent tissue and to estimate the continuous daily TCDD intake over the relevant exposure period.
- The concentration of TCDD in the blood was the best surrogate for tissue exposure, and U.S. EPA chose appropriate studies to provide data to model effects of the reference dose.
- U.S. EPA had not adequately responded to the NAS recommendation to adopt both threshold and non-threshold methods of extrapolation to account for the uncertainty of the TCDD dose-response curve.

Recently, U.S. EPA released Volume 1 of its Reanalysis of Key Issues Related to Dioxin Toxicity under the IRIS process (US EPA 2012). This volume deals with non-cancer health effects only and describes the data and analysis

used to establish an oral reference dose for TCDD. The reference dose for chronic oral exposure was 7×10^{-10} mg/kg/day and was based on two co-critical effects:

- decreased sperm count and motility in an epidemiological cohort study of men exposed to TCDD as boys due to a 1976 industrial accident in Seveso, Italy (Mocarelli et al. 2008)
- increased thyroid-stimulating hormone in newborns exposed to TCDD (Baccarelli et al. 2008)

As of December 2012, Volume 2, which deals with cancer as the endpoint, had not been released.

3.3.6. American Conference of Governmental Industrial Hygienists

The American Conference of Governmental Industrial Hygienists has a Threshold Limit Values for Chemical Substances Committee, which conducts a hazard review and risk assessment of occupational exposures and publishes its findings as *threshold limit values* for chemical substances. This committee generally follows the reviews of other agencies, if available, and then recommends threshold limit values. Their most recent evaluation listed 2,4,5-T as *not classifiable as a human carcinogen* (ACGIH 2011). TCDD was not evaluated.

3.3.7. Canadian assessments

Canadian assessments of dioxins are as follows:

- **Health Canada** concluded that TCDD may produce a range of effects in animals and humans including chloracne; liver problems; impairment of the immune, endocrine and reproductive systems; effects on the developing nervous system and other development events; and certain cancers (Health Canada 2005).
- **The Canadian Council of Ministers of the Environment** published Canadian Environmental Quality Guidelines, which said that dioxins should be considered toxic because they "can cause chloracne, fluctuations in liver enzyme levels in the blood, pulmonary deficiency, possible hepatotoxicity, as well as effects on the central and peripheral nervous system" and "although the evidence is somewhat less conclusive, some metabolic, cardiovascular, endocrine, reproductive or developmental responses may also be associated with elevated levels of exposure to PCDD/Fs" (polychlorinated dibenzodioxin/furans) (CCFME 2002).
- **Health Canada commissioned Intrinsik Environmental Sciences** to conduct a toxicological review of dioxins, which said that human populations highly exposed to TCDD may experience chloracne, increases in liver enzymes, cardiovascular disease, and developmental effects. Diabetes, thyroid dysfunction, and central and peripheral nervous system defects were other possible health outcomes (Intrinsik Environmental Sciences 2011). Evidence was inconclusive for renal, ocular, and immunological effects and birth defects. The reviewers concluded that the epidemiological evidence was stronger for all cancers combined than for any individual cancer.

3.4. Individual reviews and health hazard assessments

Individuals and groups of scientists have also reviewed the evidence for health hazards due to 2,4,5-T and TCDD exposure. These reviewers have raised issues related to interpreting toxicological and epidemiological data, including the lack of consistency of findings across studies, different characterizations and assumptions related to exposure assessment, the lack of precedent for classifying TCDD as a substance that can result in many different types of cancer, possible publication bias, an unusually long time period for cancer to develop for a substance that is not genotoxic, weaknesses in exposure assessment, and possible confounding by other cancer risk factors.

These methodological issues are relevant to evaluating all epidemiological studies and have been considered in all agency and institutional reviews, such those by IARC, U.S. EPA, NTP, and IOM. The likely occurrence and impact of these possible methodological limitations can be reviewed using established principles and procedures. Brief summaries of these reviews by individuals follow.

Johnson (1992) reviewed the literature related to TCDD and cancer and concluded that the evidence did not support a causal relationship for malignant lymphoma or soft-tissue sarcoma. He raised the following issues related to drawing a causal interpretation from epidemiological studies: TCDD exposures are higher among herbicide manufacturers than appliers, so relative risks should show a similar pattern but did not, and individuals applying phenoxy herbicides are likely exposed to other pesticides that may be responsible for or may modify the association with TCDD.

Crump et al. (2003) conducted a meta-analysis of the following occupational cohorts with exposure to TCDD and TCDD-like compounds and found a statistically significant trend for risk of total cancer mortality with increasing TCDD exposure: NIOSH cohort (Fingerhut et al. 1991), Hamburg cohort (Flesch-Janys et al. 1998), and BASF cohort (Ott and Zober 1996).

Starr (2003) commented on U.S. EPA (2003) conclusions about TCDD carcinogenicity and the meta-analysis of three cohorts by Crump et al. (2003), who had concluded that TCDD exposures of about three times background levels would lead to excess cancers among humans. Starr (2003) concluded that total exposure to TCDD-like compounds would not result in more cancer deaths and that the following accounted for his differences with U.S. EPA and Crump et al.: different selections of the dose metric, varying assumptions about the half-life of elimination, different assumptions about the contribution of the most recent 15 years of exposure, and different extrapolations of potential effects. Starr (2003) indicated that resolving the differences required detailed information on workplace exposure to TCDD and a dose-response model that considers TCDD as a cancer promoter.

Cole et al. (2003) reviewed the evidence for TCDD and cancer and concluded that TCDD is not carcinogenic to humans at low levels and may not be so at higher levels. They criticized the IARC (1997) and U.S. EPA (2000) reviews as inappropriate because:

- The conclusion that TCDD may cause multiple types of cancer has no precedent.
- The animal data are much stronger than the human findings, and large interspecies variation in dose-response slopes severely limits extrapolating from animals to humans.
- The use of the linear dose-response model is scientifically unjustified.
- Human data may be confounded by smoking and other occupational exposures.
- It is unusual for a substance to be moved to the known human carcinogen category after languishing for years in a less conclusive category.

Steenland et al. (2004) reviewed TCDD as a carcinogen. They showed that since the 1997 IARC review that classified TCDD as a human carcinogen, four new exposure-response analyses of industrial cohorts have strengthened the evidence for that classification. They also found that more information is available about how the Ah receptor could mediate TCDD's carcinogenicity.

Boffetta et al. (2011) reviewed the epidemiological literature on TCDD and cancer, concluding that the evidence does not rise to the level necessary to demonstrate a causal relationship. They found the following epidemiological weaknesses: possible publication bias, long latency shown in some studies (atypical for a promoter such as TCDD), and emphasis on all cancers combined rather than specific sites, and they suggested the association of TCDD with all cancers combined was a unique finding in cancer epidemiology.

Brody et al. (2007) reviewed the epidemiological literature on environmental pollutants (including TCDD) and breast cancer, noting that several studies reported an association but others did not. They concluded that the evidence is suggestive but that only two studies had adequately controlled for established breast cancer risk factors.

Guernsey (2007) reviewed the epidemiological literature on several herbicides used at CFB Gagetown, New Brunswick, including 2,4,5-T and TCDD. The earlier report by the IOM (2005) was the basis for this assessment, but the author conducted an independent evaluation. Guernsey agreed with the conclusions of the IOM (2005) that the scientific literature presents sufficient evidence to associate exposure to TCDD-contaminated chlorophenoxy herbicides and soft tissue sarcoma and non-Hodgkin lymphoma and limited or suggestive evidence for Hodgkin disease; myeloma; chronic lymphocytic leukemia; laryngeal, breast, and prostate cancers; spina bifida; Parkinson disease; and diabetes. The reviewer did not evaluate individual herbicides or TCDD alone.

Humblet et al. (2008) reviewed 12 cohort studies to evaluate whether risk of death from cardiovascular disease is associated with dioxin exposure. Ten studies focused on occupational or military exposures, and two on environmental exposures. The reviewers grouped studies and evaluated them by quality, based on use of an internal reference for comparison and the quality of exposure assessment. Studies using external comparisons and crude exposure assessment procedures were deemed to be of lower quality, and they showed no association between mortality from cardiovascular disease and TCDD. Higher-quality studies showed a consistent association between TCDD and increased risk of ischemic heart disease and all cardiovascular disease mortality. A meta-analysis was not conducted, but the authors concluded that TCDD exposure was associated more strongly with ischemic heart disease and less strongly with all cardiovascular disease.

Remillard and Bunce (2002) reviewed the scientific literature and concluded that except for studies of chemical manufacturing workers, TCDD exposure was associated with a consistent small increase in diabetes risk. They said that the relative risks might be underestimated because no TCDD-free population was available for comparison and actual past TCDD levels must be extrapolated, which could result in exposure misclassification. They suggested that peroxisome proliferator-activated receptor functions may be the mechanism related to development of diabetes.

Arisawa et al. (2005) concluded the evidence was unclear whether TCDD-related compounds were associated with diabetes, endometriosis, or altered thyroid function, but results were consistent for an association with defective brain development in infants.

3.5. Individual studies with exposure-response information for TCDD

A few epidemiological studies have provided exposure-response information for some outcomes. Information from these studies is presented below and in Table 3.2.

Green (1991) evaluated mortality for a cohort of 1222 forestry workers employed by Ontario Hydro between January 1, 1950 and December 31, 1982, who had possibly been exposed to phenoxy acetic acids, TCDD, and other herbicides. This cohort was also part of the IARC International Cohort Study of Phenoxy Herbicide and Chlorophenol Production Workers and Sprayers (Kogevinas et al. 1997). Cohort mortality was compared with Ontario provincial death rates through 1982. Standardized mortality rates (SMR) were not significantly elevated for any outcome: all causes—SMR = 94 (95% CI, 75-117), circulatory system—SMR = 92 (95% CI, 61-134), and all tumours combined—SMR = 109 (95% CI, 65-173). No other cause had more than 5 deaths. However, because there were only 80 deaths in the cohort, it was difficult to distinguish excesses or deficits in any cause of death category.

Table 3.2. *Exposure-response information for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) from epidemiological studies.*

Source	Study description	Outcome	Exposure	Results	
Green 1991	Ontario Hydro cohort	various diseases	phenoxy acid herbicides	• no excess for total cancer • numbers of individual cancers too small to evaluate • no excess for cardiovascular disease overall	
Kogevinas et al. 1997	36 cohorts of herbicide applicators/ producers from 12 countries	all cancer deaths combined	years of TCDD exposure	**Years** 1-4 5-9 10-19 ≥20	**Relative risk (95% CI[a])** 1.01 (0.86-1.18) 0.96 (0.79-1.17) 1.06 (0.87-1.3) 0.99 (0.75-1.2)
Vena et al. 1998	36 cohorts of herbicide applicators/ producers from 12 countries	diabetes	years of TCDD exposure	**Years** 1-4 5-9 10-19 ≥20	**Relative risk (95% CI)** 1.07 (0.39-2.94) 1.01 (0.28-3.62) 2.52 (0.89-7.11) 1.13 (0.20-6.43)
Ketchum et al. 1999	Veterans in Ranch Hand Study (980 exposed; 1275 unexposed)	total cancer	exposure categories in ppt[b]	**Exposure categories (ppt[b])** ≤10 10-94 ≥95	**Relative risk (95% CI)** 0.8 (0.5-1.3) 1.3 (0 .8-2.2) 0.7 (0.3-1.4)
Steenland et al. 2001	NIOSH cohort of TCDD exposed workers	total cancer	exposure estimates in ppt-years based on serum levels from a sample	**Exposure estimates (ppt-years)** <135 135≤520 521≤-<1212 1212-<2896 2896-<7568 7568-<=20445 =>20455	**Relative risk (95% CI)** 1.0 (referent) 1.26 (0.79-2.00) 1.02 (0.62-1.65) 1.43 (0.91-2.25) 1.46 (0.93-2.30) 1.82 (1.18-2.82) 1.62 (1.03-2.56)
Baccarelli et al. 2002	Seveso population	immune system (IgG[c] IgM[d], IgA[e], C3[f] and C4[g])	serum levels of TCDD	• IgG inversely associated (r=-0.35, p=0.0002) • IgM, IgA, C3, and C4 not associated with TCDD levels	
Hosnijeh et al. 2011	Dutch herbicide cohort	immune system (IgG, IgM, IgA, IgD[i], IgE, C3, C4, antibodies to common allergens)	serum TCDD, PCDF[h], and PCB[i] levels used to estimate current and maximum TCDD levels	• borderline association with TCDD maximum levels at C4 -0.02(-0.030–0.00)	

Source	Study description	Outcome	Exposure	Results
Viel et al. 2011	Case-control study of non-Hodgkin lymphoma in France (34 cases and 34 controls)	non-Hodgkin lymphoma	serum TCDD levels; residences near a solid waste incinerator; PCDD[l]—2,3,7,8 substituted dioxins PCDF—2,3,7,8 substituted furans	**ORs[k] per 10 ng/g lipid** PCDD 1.12 (1.03-1.26) PCDF 1.16 (1.03-1.35) DL[m]-PCB 1.04 (1.00-1.07)
Warner et al. 2011	Seveso Women's Health Study	total cancer, breast cancer	serum dioxin levels (ppt)	**Total cancer** **Serum dioxin levels (ppt)** / **Relative risk (95% CI)** 20.1- 47.0 / 1.23 (0.48 -3.16) 47.1-135.0 / 2.50 (1.02-6.09) >135 / 2.77 (1.11-6.90) **Breast cancer** **Serum dioxin levels (ppt)** / **Relative risk (95% CI)** 20.1-47.0 / 0.94 (0.28-3.14 47.1-135.0 >135 / 1.95 (0.64-5.95) > 135 / 1.98 (0.62-6.32)
Boers et al. 2012	Dutch herbicide cohort	cancer and non-cancer mortality	estimates (ppt) for cohort from serum from a sample of workers	**HR[n] increase per log TCDD unit**: Non-cancer—1.09 (1.03-1.16) Non-Hodgkin lymphoma—1.36 (1.06-1.74) Heart disease—1.19(1.08-1.32) **HR by exposure categories:** **Non-cancer** **Estimates (ppt) for cohort** / **Relative risk (95% CI)** ≤0.4 (referent) 0.4-1.9 / 1.02 (0.76-1.36) 1.9-9.9 / 1.34 (1.01-1.77) >9.9 / 1.52 (1.12-2.05) **All cancer** **Estimates (ppt) for cohort** / **Relative risk (95% CI)** ≤0.4 (referent) 0.4-1.9 / 0.67 (0.44-1.01) 1.9-9.9 / 0.76 (0.50-1.15) >9.9 / 1.32 (0.88-1.96) **Non-Hodgkin lymphoma** **Estimates (ppt) for cohort** / **Relative risk (95% CI)** ≤0.4 (referent) 0.4-1.9 / 2.99 (0.21-43.29) 1.9-9.9 / 5.28 (0.48-58.06) >9.9 / 10.28 (1.05-5.95) **Urinary tract cancers** **Estimates (ppt) for cohort** / **Relative risk (95% CI)** ≤0.4 (referent) 0.4-1.9 / 0.76 (0.23-2.57) 1.9-9.9 / 1.32 (0.45-3.81) >9.9 / 2.04 (0.70-5.95)

[a] *CI = confidence interval.*
[b] *ppt = parts per trillion.*
[c] *IgG = immunoglobulin G.*
[d] *IgM =immunoglobulin M.*
[e] *IgA = immunoglobulin A.*
[f] *C3 = component 3.*
[g] *C4 = component 4.*
[h] *PCDF = polychlorinated dibenzyl furan.*
[i] *IgD = immunoglobulin D.*
[j] *PCB = polychlorinated biphenyl.*
[k] *OR = oral reference.*
[l] *PCDD = polychlorinated dibenzyldioxin.*
[m] *DL = dioxin-like.*
[n] *HR = hazard ratio.*

For the IARC International Cohort Study of Phenoxy Herbicide and Chlorophenol Production Workers and Sprayers, which included 36 cohorts in 12 countries, Kogevinas et al. (1997) evaluated the mortality of workers producing or using phenoxy herbicides or chlorophenols. They estimated exposure from individual job records and the availability of TCDD and other dioxin and furan measurements from some cohorts. The relative risk for mortality for all cancer combined was 1.29 (0.94-1.78) among those exposed to TCDD or higher chlorinated dioxins compared with unexposed workers. Relative risks for total cancer were not associated with exposure duration. Relative risks were:

- 1-4 years—1.01 (0.86-1.18)
- 5-9 years—0.96 (0.79-1.17)
- 10-19 years—1.06 (0.87-1.30)
- ≥20 years—0.99 (0.75-1.2)

Relative risk by years since first exposure were:

- 0-9 years—1.0
- 10-19 years—1.04 (0.84-1.30)
- ≥20 years—1.26 (0.99-1.60)

For an analysis of deaths other than cancer in the IARC International Cohort Study, Vena et al. (1998) found the following related to TCDD:

- TCDD was not associated with cerebrovascular disease.
- Ischemic heart disease mortality was elevated among the exposed (1.67 (1.23-2.26)).
- No trend for ischemic heart disease occurred with duration of exposure or years since first exposure.
- Non-significant excesses were observed for cerebrovascular disease among exposed individuals (1.54 (0.83-2.88)) and diabetes (2.25 (0.53-9.50)).
- The risk of diabetes increased by years since first exposure (p for trend = 0.18):

  10 to 19 years—2.34 (0.56-9.83)
  20 or more years—1.54 (0.30-7.82)

- Risk for diabetes increased by duration of exposure:

  1 to 4 years—1.07 (0.39-2.94)
  5 to 9 years—1.01 (0.28-3.62)
  10 to 19 years—2.52 (0.89-7.11)
  20 or more years—1.13 (0.20-6.43)

- Cerebrovascular disease was not associated with years since first exposure or exposure duration.

Ketchum et al. (1999) conducted a study of 980 Vietnam veterans included in the Operation Ranch Hand Study, as well as 1275 other Air Force veterans. They found no exposure-dependent relationship with total cancer or with cancers of the skin, eye, ear, face, head/neck, oral cavity, pharynx/larynx, kidney/bladder, prostate, or lung. Serum TCDD levels were categorized as background (≤10 ppt), low (>10 to ≤94 ppt), and high (≥95 ppt). The oral references for total cancer were 0.8 (0.5-1.3) for background, 1.3 (0.8-2.2) for 10-94 ppt, and 0.7 (0.3-1.4) for ≥95 ppt. No grouping of cancers showed an exposure-response pattern, and analyses stratified by <20 years or ≥20 years after service in Vietnam showed no exposure-response relationship with total cancer or any specific type. The panel notes that this is a small cohort study with the power to detect only very large relative risks.

Steenland et al. (2001) evaluated total cancer mortality among 3538 workers from 12 plants that produced TCDD-contaminated products (Fingerhut et al. 1991). Serum TCDD levels from 199 workers in one plant were used to

estimate a TCDD level for all workers based on modelling to develop exposure scores. Rate ratios for septile categories of estimated cumulative serum levels (lagged 15 years) by ppt-years were:

- <135 (1.0–referent)
- 135≤520 (1.26 (0.79-2.00))
- 520≤1212 (1.02 (0.62-1.65))
- 2,896≤7,568 (1.46 (0.93-2.30))
- 7,568≤20,455 (1.82 (1.18-2.82))
- >20,455 (1.62 (1.03-2.56))

Baccarelli et al. (2002) evaluated the immunological response by TCDD levels in a sample of the Seveso population (in Italy) exposed to high TCDD levels due to an industrial accident. Plasma immunoglobulin (IgM, IgA) and complement component (C3, C4) concentrations were not associated with TCDD levels, but IgG levels were inversely associated (r= - 0.35, p=0.0002).

Pesatori et al. (2009) evaluated cancer incidence after 20 years of followup of individuals exposed to TCDD from the Seveso accident. Exposure was based on TCDD measurements taken in zones surrounding the accident site. Zone A was the most heavily contaminated, Zone B had medium exposure, and Zone R had low exposure. Non-contaminated surrounding municipalities provided referent rates. Findings were as follows:

- Compared with the non-contaminated, referent areas, total cancer in the contaminated zones was 0.96 (0.91-1.00) for low exposure (Zone R), 1.00 (0.89-1.13) for medium exposure (Zone B), and 1.03 (0.76-1.38) for high exposure (Zone A).
- No individual cancer showed statistically significant trends with exposure, although non-significant excesses in the high exposure (Zone A) occurred for breast cancer (1.43 (0.71-2.87)) and uterine cancer (2.34 (0.87-6.27)).
- Lymphatic/hematopoietic cancer was elevated for high (Zone A) (1.39 (0.52-3.71)) and medium (Zone B) (1.56 (1.07-2.27)) exposures.
- Relative risks were larger 15 or more years since the accident in Zone A (high exposure) for lymphatic/hematopoietic cancer (2.96 (0.95-9.22)) and breast cancer (2.57 (1.07-6.20)) but not for total cancer (1.27 (0.81-2.00)).

Warner et al. (2011) evaluated cancer risks among women in the Seveso Women's Health Study. Serum TCDD levels were available for 835 women, who were asked if they had been diagnosed with cancer. Medical records were obtained to verify self-reporting. Total cancer and breast cancer were evaluated in relation to serum TCDD levels, adjusted for several potential confounding factors. Results were as follows:

- For all cancers combined, the adjusted hazard ratios by TCDD ppt were as follows (where p for trend = 0.002, compared with TCDD levels ≤20.0 ppt):

  1.23 (0.48-3.16) for 20.1 to 47.0 ppt
  2.50 (1.02-6.09) for 47.1 to 135.0 ppt
  2.77 (1.11-6.90) for >135.0 ppt

- For breast cancer, the adjusted HRs were (p for trend = 0.09):

  0.94 (0.28-3.14) for 20.1 to 47.0 ppt
  1.95 (0.64-5.95) for 47.1 to 135.0 ppt
  1.98 (0.62-6.32) for >135.0 ppt

- Hazard ratios for all cancer combined and for breast cancer varied little by length of followup.

Boers et al. (2012) used plasma TCDD levels from a sample of workers to predict levels in the entire Dutch herbicide cohort based on a detailed occupational history. A Cox proportional hazards model was used to evaluate the relationship between TCDD levels and mortality. Analyses were based on log-transformed TCDD estimates lagged one year. Their findings were as follows:

- Hazard ratios for each TCDD unit increase (ppt) on the log scale were:

  for non-cancer mortality—1.09 (95% CI 1.03-1.16)
  for non-Hodgkin lymphoma—1.36 (95% CI 1.06-1.74)
  for ischemic heart disease—1.19 (95% CI 1.08-1.32)

- Exposure categories of ≤0.4 (referent), 0.4 to 1.9 (low), 1.9 to 9.9 (medium), and ≥ 9.9 (high) showed the following hazard ratios:

  all non-cancer causes of death—1.02 (0.76-1.36), 1.34 (1.01-1.77), and 1.52 (1.12-2.05)
  all cancer deaths—0.67 (0.44-1.01), 0.76 (0.50-1.15), and 1.32 (0.88-1.96)
  non-Hodgkin lymphoma—2.99 (0.21-43.29), 5.28 (0.48-58.06), and 10.28 (1.05-5.95)
  urinary tract cancers—0.76 (0.23-2.57), 1.32 (0.45-3.81), 2.04 (0.70-5.95)

3.6. Issues in interpreting epidemiological studies

Reviewers of the epidemiological literature on 2,4,5-T and TCDD have raised some common concerns about interpreting study findings. Following are the panel's comments about these and other methodological issues in epidemiology.

3.6.1. Exposure assessment

Exposure assessment is critical for all epidemiological studies. High-quality assessment is essential to adequately compare the risk of exposed and unexposed populations for any disease and to understand exposure-risk relationships. Exposure assessment is particularly challenging in observational epidemiology studies. The lower the actual level of human exposure, the more critical it is to have an accurate exposure assessment, and the more difficult this accuracy is to achieve. Even the most comprehensive and high-quality epidemiological studies typically have monitoring information on exposure for only a few points in time for some of the study population, which must be used to represent the exposure history of the entire population.

Thus, for much of the timeframe covered by any epidemiological study, exposures must be estimated, or extrapolated, from a relatively small amount of monitoring data, along with other useful determinants of exposure. Regardless of the sophistication of the extrapolation process, errors in exposure assessment are unavoidable. A major challenge in assessing epidemiology studies is to gauge the relative magnitude and direction of exposure misclassification—and how exposure misclassification might influence relative risk estimates.

Sometimes false positive associations are presented as the only consequence of exposure misclassification (Boffetta et al. 2008). However, exposure misclassification can lead to an underestimation of the relative risk and to false negative conclusions (Blair et al. 2009). Exposure misclassification can either be *non-differential* or *differential* (Checkoway et al. 2004), with different effects on relative risk estimates.

For non-differential exposure misclassification, the degree of misclassification is the same for diseased and non-diseased populations. Estimates of relative risks tend to be biased towards the null and to create false negative findings. This type of misclassification occurs in every epidemiological study, even those with the highest-quality exposure assessments.

For differential exposure misclassification, the scope or size of the exposure misclassification differs for the diseased and non-diseased. Estimates of relative risk can be biased either towards or away from the null, depending on the direction of errors related to disease status (Checkoway et al. 2004). This type of misclassification is of greatest concern with studies that involve exposure information from individuals who know their disease status, such as case-control or cross-sectional studies. It is unlikely in cohort studies, where exposures or information used to characterize exposures are typically taken from information obtained before the disease developed. Typically, exposure misclassification results in assigning exposures that are too high for some individuals and too low for others. It flattens any true exposure-response gradient and leads to false negative, rather than false positive, conclusions (Blair et al. 2009).

With any study, it is helpful to gauge the likely direction and magnitude of misclassification effects on relative risk estimates. This step is important not only for assessing how misclassification could affect estimates of relative risk but also for comparing those possible effects with other biases (e.g., confounding). Evaluating the overall strength of a study often requires balancing different (and sometimes opposite) weaknesses and biases.

If an exposure and an outcome have no underlying association, non-differential misclassification cannot by itself create a false positive association. However, differential misclassification could create a false positive association when no underlying relationship exists because the degree of exposure misclassification is related to disease status. Differential misclassification could also diminish a true association, depending on the direction of the misclassification relative to disease status.

Another limitation of epidemiological studies of 2,4,5-T and TCDD is that people are exposed to both at once, so it is difficult to separate their effects. However, their long-term exposure patterns may differ, because the half-life of TCDD in the environment and human tissues is measured in years, while that of 2,4,5-T is measured in days. TCDD's longer half-life allows more potential use of biological measures for exposure assessment. Measurements of 2,4,5-T are typically air or skin measurements and must be taken at time of use.

Although the exposure assessment quality for TCDD is probably superior to that for 2,4,5-T, the possibility of exposure misclassification is still considerable, even with studies that have some biological measures for TCDD exposure. Studies with individual biological measures typically have only one or a few measurements per person, and they may not be from the toxicologically critical time period. In addition, the half-life of these substances in humans varies considerably, and extrapolating to previous body burdens from a later measurement is challenging. Although TCDD exposure estimates have been made for several epidemiological studies, they are not done without error, and even a modest amount of misclassification can greatly affect relative risk estimates. For example, a 30% non-differential misclassification of an exposure would result in a calculated relative risk of 1.15 for a true relative risk of 2.00 and 1.31 for a true relative risk of 3.00 (Blair et al. 2007). These examples show that relatively modest exposure misclassification can lead to a calculated relative risk that is small enough to—depending on other circumstances—lead the investigator to conclude that an association does not exist.

Another example that shows the possible magnitude of exposure misclassification: When the correlation between exposure estimates and true exposure is 0.70, which is very high for epidemiological studies, only 40% of the

subjects would be placed in the correct quintile of exposure study (Walker and Blettner 1985). This finding is sobering because the accuracy level of exposure assessment in occupational studies is probably closer to 40%—or even less (Blair et al. 2007). If the typical accuracy of exposure estimates is only about 40%, estimates of relative risks would likely be less than half their true value.

Clearly, exposure misclassification can and probably does have a powerful effect in nearly all observational epidemiology studies. What's more, it typically drives relative risk estimates towards the null.

Misclassification of disease has the same effect on relative risk estimates as misclassification of exposure. Although some disease misclassification occurs in many epidemiological studies, it is usually much less than that for exposure. However, the degree of misclassification varies considerably by disease. Typically, diagnoses of cancer based on medical records are accurate, but diagnoses based on death certificates are less so and vary considerably by cancer type (Percy et al. 1981). Self-reports are sometimes used to determine cancer, and they are even more prone to error than use of death certificates. Most non-malignant diseases follow the same pattern, with the accuracy of diagnosis highest when based on medical records, lower when based on mortality records, and lowest when based on self-reports.

3.6.2. Confounding

In observational epidemiology, biases can occur because exposure is not assigned experimentally, resulting in differences in disease risk factors (i.e., diet, geographic location, personal habits, and personal and family disease history) by exposure among the study population. This issue is not related to quality of exposure assessment but is a concern because these factors may be non-randomly distributed among the exposed and unexposed populations. If these factors are associated with both the disease and the exposure of interest, confounding can occur, which may bias estimates of relative risks away from or towards the null. In observational epidemiological studies, study design and analytic techniques are used to control for such possible biases because it's not possible to randomly assign exposure to study participants.

The possibility of confounding must be considered in all epidemiological studies. Confounding occurs when a risk factor for the outcome or disease of interest is also associated with the exposure under study (Checkoway et al. 2004). Confounding can lead to a false association between an exposure and a disease, when a positive association exists between the confounding factor and the disease. Confounding can also hide an association if the confounder (e.g., some dietary micronutrients) prevents the disease and is also associated with the exposure of interest. However, for a confounder to have much of an effect, it must be as strong a risk factor for the disease as is the exposure of interest and it must be tightly correlated with the exposure of interest (Breslow and Day 1980).

Various approaches are used to control confounding. Matching of study subjects on potential confounders is a design approach. For example, if a study of an occupational exposure and lung cancer included only non-smokers (i.e., matched on non-smoking status), then tobacco use could not be a confounder because no tobacco users were included in the study. Matching is commonly used in case-control and cross-sectional studies.

With the analytic approach to control confounding, information on potential confounders is obtained for each subject. With such information, confounding can be controlled, or managed, by using a statistical approach to remove the influence of possible confounding effects on estimates of relative risk by calculating relative risks within various strata of the confounder. The analytic approach is used in nearly all epidemiological investigations, whether or not matching is used.

However, even when information is obtained on a number of possible confounders, confounding could still occur due to some factor that has not been controlled by study design and for which no information is available to make a statistical adjustment. Even in these situations, it is feasible to assess the possibility of confounding and its strength, if it occurs. First, the scientific literature can be surveyed for evidence that the suspected confounder is actually associated with the disease or outcome of interest and if it is associated, the level of observed relative risk. If the literature suggests there is no association between the putative confounder and outcome of interest, then it is reasonable to conclude that no confounding occurs. If there is evidence that the possible confounder is associated with the outcome of interest, the relationship must be as strong as that between the disease and the exposure of interest, or the confounder cannot fully explain the study finding (Pearce and Greenland 2005). The possible confounder and the exposure of interest must also be correlated.

It is often difficult to obtain direct information about the association between two possible risk factors. However, even if no empirical information is found in the scientific literature, it is possible to judge whether such an association is likely to exist and thus to assess the possible magnitude of confounding (Blair et al. 2007).

The healthy worker effect is a form of selection bias that may occur in some cohort studies. It is mainly a concern in retrospective cohort studies for which the mortality of an occupational cohort is compared with the mortality of the general population (Checkoway et al. 2004). This type of comparison often leads to an underestimate of the relative risk because members of the occupational cohort must be healthy to be working, which is not the case for the general population. Thus, occupational cohorts typically have lower total mortality rates than the general population, as well as lower mortality for some diseases. Despite this limitation, the general population is often used as the comparison population and to generate expected numbers for a non-exposed population because it is large; relevant disease information is readily available; and often no unexposed working population is readily available. The healthy worker bias can be largely avoided by choosing active workers who have not been exposed to the chemical of concern as the comparison group (Checkoway et al. 2004), which is often achieved by comparing exposed and unexposed subgroups within the cohort.

The panel concluded that agency and institutional reviews of 2,4,5-T and TCDD have adequately considered possible limitations of epidemiological studies. Confounding is controlled adequately in enough studies to conclude it is likely not responsible for the association between TCDD and various human health outcomes. Errors in TCDD exposure estimates undoubtedly occur. The quality of exposure assessment is probably as good for TCDD as for many other established human health hazards because this chemical persists in the environment and in human tissues. Exposure misclassification of TCDD would more likely reduce estimates of relative risk rather than increase them.

Information available on exposure to the active compound in 2,4,5-T is limited and cannot be separated from exposures to TCDD.

3.7. Experimental animal studies

The panel considered the implications of animal studies for humans exposed to 2,4,5-T and TCDD (Table 3.1). Human health concerns emerging from experiments using laboratory animals include reproductive and endocrine dysfunction, suppression of immune responses, neurological effects, cardiovascular effects, and cancer. These health concerns primarily stem from extensive studies on TCDD toxicity in laboratory animals; data on the toxicity of pure 2,4,5-T (independent of TCDD) are limited.

Since 1994, NAS expert committees have extensively reviewed the scientific literature on 2,4,5-T and TCDD toxicity and published biennial updates under the title Veterans and Agent Orange. Health Effects of Herbicides used in Vietnam (e.g., IOM 1994, 2005, 2012). These biennial updates were a major source of information cited in this section. The U.S. EPA Dioxin Reassessment Update 2012 (US EPA 2012) also provided a useful compendium of toxicity studies that met high-quality criteria for use in risk assessment and on which the panel relied. The following subsections present a concise overview of the known toxicity of 2,4,5-T and TCDD.

3.7.1. Metabolism

The body's ability to absorb, metabolize, and excrete a chemical is an important factor in determining toxicity. Both 2,4,5-T and TCDD are readily absorbed into the bloodstream following oral and inhalation exposure, while skin absorption is much lower. However, 2,4,5-T is water-soluble and readily excreted from the body, while TCDD is fat-soluble, poorly metabolized, and excreted slowly. The fact that 2,4,5-T clears rapidly from the body greatly reduces the likelihood of chronic toxicity, while TCDD can accumulate in body tissues when intake exceeds excretion. Thus, it is possible to achieve toxic levels of TCDD over time due to chronic low-level exposure.

However, TCDD's slow metabolic rate can be increased if the person is also exposed to compounds that induce enzymes that metabolize TCDD, such as the polycyclic aromatic hydrocarbons in cigarette smoke. In fact, smoking has been associated with a 30% decrease in TCDD half-life (Flesch-Janys et al. 1998).

Once absorbed, TCDD associates primarily with the lipoprotein fraction of blood and later moves into tissues throughout the body. The liver and fatty tissues are the primary storage sites. TCDD concentration in the lipid fraction of blood serum has been shown to be in equilibrium with the lipid fraction in other tissue compartments. The lipid-adjusted blood serum concentration of TCDD is often used to estimate total body burdens.

3.7.2. Acute toxicity

2,4,5-T is classified as a compound of moderate acute toxicity following a single exposure, with an oral LD50 (lethal dose sufficient to kill 50% of test population) of 389 mg/kg of body weight in mice and 500 mg/kg in rats. Death from acute poisoning has been attributed to mitochondrial toxicity and the uncoupling of oxidative phosphorylation, a vital process that cells use to generate energy. Death from a lethal dose of 2,4,5-T is rapid and due to multiple organ failure.

TCDD is classified as having extremely high acute toxicity even though death does not occur immediately following a single dose. The toxic response to a lethal dose is unique and is referred to as a *wasting syndrome;* Animals slowly lose weight over one to three weeks (depending on species), with reduced appetite, loss of body fat stores, liver damage, and atrophy of the thymus gland. Biochemical effects include inhibition of glucose production and activation of the enzyme *lipoprotein lipase.* The lethal dose of TCDD varies widely, with guinea pigs being the most sensitive (LD50 ≈ 1 µg/kg) and hamsters the most resistant (LD50 ≈ 5000 µg/kg). The LD50 also varies within species, as has been shown with different inbred mouse and rat strains. The LD50 of TCDD in rhesus monkeys (*Macaca mulatta*) is about 70 µg/kg. The LD50 in humans is not known; a serum level of 108 µg/kg TCDD was found in Victor Yushchenko, who survived intentional TCDD poisoning (Sorg et al. 2009). The basis for the wide variation in lethal TCDD dose among species has not been determined.

3.7.3. Organ-specific chronic toxicity

Repeatedly exposing laboratory animals to TCDD at doses below those causing acute toxicity (e.g., body weight loss) has been shown to adversely affect many tissues and organs in the body, including the liver, heart, and skin, as well as the immune, endocrine, and reproductive systems. In addition, when pregnant females are fed or injected with TCDD, a spectrum of birth defects are observed. In contrast, there is little evidence of chronic toxicity following exposure to pure 2,4,5-T. Following is a summary of notable findings for both chemicals.

3.7.3.1. Reproductive and developmental toxicity

Several studies have reported that 2,4,5-T can be developmentally toxic in rodents, resulting in increased fetal mortality and malformations. These effects generally occurred at doses greater than 20 mg/kg, when administered to the pregnant female on days 6-15 of gestation. However, in most of these studies, 2,4,5-T purity was not known or not reported. 2,4,5-T was fetotoxic to mice, retarding growth and causing increased embryo death and cleft palate. No comparable developmental effects were seen in rabbits, sheep, or monkeys treated with 2,4,5-T during pregnancy. The most reliable study on the reproductive and developmental toxicity of 2,4,5-T was published by Smith et al. (1981). In this three-generation feeding study in female rats exposed to 0, 3, 10 or 30 mg/kg 2,4,5-T containing <0.05 ppm TCDD (Table 3.3), the only adverse outcome was reduced survival of full-term pups after birth. The lowest exposure level causing this effect was 10 mg/kg. Based on these data, a reference dose of 0.01 mg/kg 2,4,5-T was calculated using a 1000-fold safety factor for extrapolation from animals to humans.

Table 3.3. *Results of studies of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) toxicity in laboratory animals.*[a]

Source	Species	Protocol	Results	No observed adverse effect level (mg/kg/day)	Lowest observed adverse effect level (mg/kg/day)	Reference dose (mg/kg/day)
Bionetics 1968 Innes et al. 1969	Mice	21.5 mg/kg by gavage[b] beginning at age 7; same amount unadjusted for body weight daily until age 28 days; then 60 mg/kg in diet until age 78 weeks	no tumours no other effects			
Bionetics 1968 Innes et al. 1969	Mice	215 mg/kg single dose by gavage[b] at age 28 days; maintained to age 78 weeks	no tumours no other effects			
Collins and Williams 1971	Hamsters	Oral dosing on days 6-10 of pregnancy	malformations		100	
Muranyi-Kovacs et al. 1976	Mice	100 mg/L drinking water for 2 months, followed by 80 mg/kg in diet for life	no tumours no other effects			
Kociba et al. 1979	Rats	0, 3, 10, or 30 mg/kg/day in the diet for 2 years	no tumours increase in urinary coproporphyrins	3	10	0.01
Smith et al. 1981	Rats	3-generation feeding study at 0, 3, 10, or 30 mg/kg/day	Reduced neonatal survival	3	10	0.01

[a] *Blank cells = information not available.*
[b] *Gavage = stomach tube feeding.*

TCDD is a potent developmental toxicant, producing malformations and fetotoxic effects in embryos of all tested species:

- In pregnant rats, TCDD doses of 0.5 µg/kg/day caused decreased maternal weight gain and severe maternal toxicity. Doses as low as 0.03 µg/kg/day caused embryo death and intestinal bleeding.
- In mice, in addition to increased mortality and reduced growth, common TCDD effects on fetuses included cleft palate and hydronephrosis (kidney malformation).
- In rats, the developing male and female reproductive organs appear to be among the most sensitive to TCDD (Table 3.4). Male offspring exposed to TCDD in utero showed changes in testicular, epididymal (sperm duct), and seminal vesicle weight and function and decreased sperm production at a human equivalent dose of 0.028 ng/kg/day (Table 3.4). These effects were thought to be due to changes in testicular steroid production, which depends on a critical exposure window during fetal development.

Recently, the U.S. EPA established a reference dose of 0.7 pg/kg body weight/ day for non-cancer effects of chronic oral exposure to TCDD.

- Decreased sperm count and motility have also been observed in men exposed to TCDD as boys due to an industrial accident in Seveso, Italy (Mocarelli et al. 2008). Recently, the U.S. EPA used this finding to establish a reference dose of 7 x 10e-10 mg/kg/day for non-cancer effects of chronic oral exposure to TCDD. With an uncertainty factor of 300, the reference dose based on decreased sperm production in rats was even lower, at 5.4 x 10e-11 mg/kg/day (Table 3.4).

Table 3.4. *Candidate-recommended reference doses for the most sensitive endpoints of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxicity: Dose-response modelling by the U.S. EPA (2012) for adverse effects related to male and female reproductive system, thyroid gland, nervous system, and immune system using blood-concentration-based human equivalent dose. Modified from U.S. EPA (2012).*[a]

Source	Species, strain	Protocol[b]	Endpoint	Administered dose LOAEL[c] (ng/kg/day)	Human-equivalent dose $LOAEL_{HED}$[d] (ng/kg/day)	UF[e]	Reference dose (mg/kg/day)
Sparschu et al. 1971	Rat, Sprague-Dawley (S-D)	gavage GD[f] 6-15 n^g = 4–129	decreased fetal body weight	5.00E+02	1.7E+00	30	1.1E–08
Vos et al. 1973	Guinea pig, Hartley	8-week gavage n = 10	decreased delayed-type hypersensitivity response to tuberculin	5.71E+00	3.2E–02	30	2.1E–10
Murray et al. 1979	Rat, S-D	3-generation dietary	reduced fertility and neonatal survival— F0 (parent) F1 (offspring)	1.00E+01	3.8E–01	30	9.6E–10
Toth et al. 1979	Mouse, Swiss/H/Riop	1-year gavage n = 38-44	dermal amyloidosis skin lesions		9.9E–03	300	3.3E–11
DeCaprio et al. 1986	Guinea pig, Hartley	90-day dietary n = 10	decreased body weight organ weight changes (liver, kidney, thymus, brain)	4.90E+00	3.3E–02	30	1.4E–10
White et al. 1986	Mouse, B6C3F1	14-day gavage n = 6–8	decreased serum complement	1.00E+01	2.8E–02	300	9.2E–11
Bowman et al. 1989a,b	Rhesus monkey	dietary exposure, 3.5-4 years n = 3-7	neurobehavioral effects	6.07E-01	8.2E-03	300	2.7E-11
Seo et al. 1995	Rat, S-D	gavage GDs[f] 10-16 n = 10	decreased serum T4[h] and thymus weight	1.00E+02	9.1E-01	30	5.6E-09
Sewall et al. 1995	Rat, S-D	30-week gavage n = 9	decreased serum T4	3.57E+01	1.7E+00	30	6.0E-09
VanBirgelen et al. 1995	Rat, S-D	13-week dietary n = 8	decreased liver retinyl palmitate	4.69E+01	5.1E-01	300	1.7E-09
Schantz et al. 1996	Rat, S-D	gavage GDs 10-16 n = 80–88	maze errors (facilitatory effect)	1.2E-01	1.7E-01	300	5.7E-10
Li et al. 1997	Rat, S-D (22 day-old female)	single gavage n = 10	increased serum follicle stimulating hormone	1.00E+01	1.7E-02	30	9.7E-11
Amin et al. 2000	Rat, S-D	gavage GDs 10-16 n = 10	reduced saccharin consumption and preference		1.7E-01	300	5.7E-10
Franc et al. 2001	Rat, Long-Evans	22-week gavage n = 8	increased relative liver weight; decreased relative thymus weight	3.00E+01	1.4E+00	30	8.7E-09
Kattainen et al. 2001	Rat, Line C	gavage GD 15 n = 4-8	inhibited molar development in pups		9.0E-02	300	3.0E-10

Source	Species, strain	Protocol[b]	Endpoint	Administered dose LOAEL[c] (ng/kg/day)	Human-equivalent dose $LOAEL_{HED}$[d] (ng/kg/day)	UF[e]	Reference dose (mg/kg/day)
Markowski et al. 2001	Rat, Holtzman	gavage GD 18 n = 4-7	neurobehavioral effects in pups (running, lever press, wheel spinning)	2.00E+01	5.2E-02	300	1.7E-10
Ohsako et al. 2001	Rat, Holtzman	gavage GD 15 n = 5	decreased anogenital distance in male pups	5.00E+01	1.8E-01	30	9.1E-10
Kuchiiwa et al. 2002	Mouse, ddY	maternal 8-week gavage before mating n = 3	F1 (first generation) male offspring: decreased serotonin-immunoreactive neurons in raphe nuclei of F1 male offspring	7.00E-01	2.7E-03	300	9.2E-12
Latchoumy-candane and Mathur 2002	Rat, Wistar	45-day oral n = 6	decreased sperm production	1.00E00	1.6E-02	300	5.4E-11
Crofton et al. 2005	Rat, Long-Evans	4-day gavage n = 4–14	decreased serum T4	1.00E+02	7.4E-01	30	5.6E-09
Franczak et al. 2006	Rat, S-D	gavage GD 14, 21, post-natal days 7, 14 n = 7	abnormal estrous cycle	7.14E+00	3.2E-01	300	1.1E-09
Li et al. 2006	Mouse, NIH	gavage GDs 1-3 n = 10	pregnant females: decreased progesterone, increased estradiol	2.00E+00	1.6E-03	300	5.3E-12
Miettinen et al. 2006	Rat, Line C	gavage GD 15 n = 3-10	pups: cariogenic lesions		8.9E-02	300	3.0E-10
Bell et al. 2007	Rat, CRL:WI (Han)	17-week dietary n = 30	puberty onset delayed	8.00E+00	8.9E-02	30	1.4E-09
Ishihara et al. 2007	Mouse, ICR	gavage weekly for 5 weeks n = 42-43	decreased male/female sex ratio	1.00E+02	5.0E-01	300	1.7E-09
Shi et al. 2007	Rat, S-D	11-month gavage n = 10	decreased serum estradiol	7.14E-01	2.7E-02	30	1.6E-10
Hutt et al. 2008	Rat, S-D	13-week dietary n = 3	embryotoxicity	7.14E+000	2.5E-01	300	8.4E-10
Keller et al. 2008a,b; 2007	Mouse, CBA/J and C3H/HeJ	gavage GD 13 n = 23-36 (pups)	pups: missing molars, changes in lower jaw shape		9.9E-03	300	3.3E-11
Smialowicz et al. 2008	Mouse, B6C3F1	90-day gavage n = 8-15	decreased SRBC[h] response	1.07E+00	6.3E-03	300	2.1E-11

[a] Blank cells = information not available
[b] Gavage = stomach tube feeding.
[c] LOAEL = lowest observed adverse effect level.
[d] LOAELHED = lowest observed adverse effect level (human equivalent dose).
[e] UF = uncertainty factor.
[f] n = number of samples.
[g] GD = gestation days.
[h] T4 = thyroxine.
[i] SRBC = sheep red blood cells.

A recent study showed that exposing pregnant rats (F0; parent generation) to TCDD resulted in expected changes in offspring (F1 generation) but unexpected changes in the F3 (grandchildren) generation, even though the F2 and F3 generations had no direct exposure to the chemical (Manikkam et al. 2012). (F2 was not evaluated.) F1 and F3 rats had a higher rate of multiple diseases. Male F3 rats had increased kidney disease, and females had increased polycystic ovary disease, as well as fewer primordial ovarian follicles. These effects were attributed to the F1 rats experiencing genetic changes in their developing sex organs while in utero and the offspring inheriting these changes. This study raises concern for possible similar effects in humans. However, the TCDD dose that Manikkam et al. (2012) used was higher than that to which humans are typically exposed, and the exposure route was experimental (injection of the pregnant rat on specific days of gestation). Thus, the direct relevance of these findings to humans is unclear.

Thyroid disease: TCDD has been shown to alter the circulating levels of thyroid hormones, including triiodothyronine (T3) and thyroxin (T4), as well as thyroid stimulating hormone (TSH), produced by the pituitary gland. In most studies, chronic TCDD exposure is associated with a hypothyroid state, with decreases in T3 and T4 and increases in TSH. The T4 reduction is so significant that it is considered a biomarker of exposure to dioxin-like compounds (Yang et al. 2010). Reference doses for TCDD have been calculated based on decreased serum T4 (Table 3.4). Even lower reference doses have been calculated based on human data showing increased TSH levels in newborns exposed to dioxins in utero (Baccarelli et al. 2008; Table 3.4). Long-term treatment of female rats with low doses of TCDD led to changes in thyroid tissues, with abnormal growth in follicular cells and follicle enlargement. These changes are consistent with TSH overstimulation of the thyroid, which is associated with thyroid cancer.

Neurotoxicity: Neurological disorders due to chemical exposure have both immediate and delayed effects. Changes in thinking, consciousness, or attention may occur with abnormalities in movement, depending on which area of the brain is damaged. Neurological injury from chemicals can include Parkinson disease, Alzheimer's disease, spinocellular (type of skin cell) degeneration, and amyotrophic lateral sclerosis (ALS).

Although the Institute of Medicine's Agent Orange committee determined that "limited, suggestive evidence" links exposure to phenoxy herbicides to Parkinson disease (IOM 2012), no animal studies show 2,4,5-T is neurotoxic. Furthermore, studies of TCDD exposure in adult animals have identified few (if any) neurotoxic effects.

However, tissue culture studies have identified plausible mechanisms of neurotoxicity that could link TCDD exposure to neurological problems. Effects include increased production of inflammatory mediators (chemicals released by irritated tissues) and increased oxidative stress, which may play a role in neurodegeneration. Such findings provide a measure of biological plausibility given that TCDD residues can be found in the brains of exposed animals. Several studies have shown that prenatal exposure to TCDD has neurotoxic effects, influencing offspring behaviour and impairing learning (Table 3.4). The neurobehavioral effects in rats are among the most sensitive adverse effects of TCDD (Table 3.4). Studies of female monkeys exposed to low levels of TCDD during pregnancy have shown changes in social/maternal behaviour that could indirectly affect neurobehaviour of offspring (Table 3.4).

Immunotoxicity: Many studies provide evidence of TCDD's immunotoxicity (IOM 2012). In mice, a single TCDD dose in the low µg/kg range suppressed both antibody- and T-cell-mediated immune responses to many antigens. TCDD exposure also increased the mortality rate and/or severity of symptoms in mice or rats infected with bacterial and viral agents, including salmonella, listeria, influenza, and herpes Type 2.

Interestingly, TCDD's immunosuppressive effects include reducing allergic responses and suppressing symptoms associated with autoimmune diseases, which have been observed in animal models of Type 1 diabetes, multiple sclerosis, and ulcerative colitis.

In 2006, the NRC agreed with the U.S. EPA that TCDD is likely to be a human immunotoxicant at "some dose level" despite the lack of consistent findings for immune system effects in humans even when exposed to relatively high levels of dioxin.

Cardiovascular effects: In 2010, the IOM Agent Orange committee reviewed epidemiological evidence that led them to conclude that "limited, suggestive evidence" links Agent Orange exposure to increased risk of hypertension and ischemic heart disease (IOM 2012). This conclusion was supported by new animal studies that showed TCDD induces hypertension and promotes cardiovascular disease development in several different animal models. The endothelial cells that line blood vessels are a known TCDD target. Exposing blood vessels in a live animal or endothelial cells grown in culture to TCDD results in many changes that have been linked to cardiovascular disease, including increased oxidative stress, production of inflammatory markers, and structural remodelling. These data provide biological evidence that TCDD exposure could increase the risk of cardiovascular disease in people.

Carcinogenicity: Laboratory animal studies on 2,4,5-T reveal a lack of carcinogenic activity even when the animals are treated for their lifetime with relatively high doses (Table 3.5). These data support the conclusion that 2,4,5-T is not a human carcinogen. However, treating rats and mice with very low doses of TCDD over their lifetimes resulted in increased incidence of several cancers, including liver, lung, adrenal gland, oral cavity, and thyroid gland. TCDD also increased the growth of tumours caused by other known carcinogens. These findings are consistent with epidemiological studies and support the conclusion drawn by several scientific committees that TCDD is a multisite human carcinogen.

Table 3.5. *Studies selected for cancer dose-response modelling by the U.S. EPA (2012).*

Reference	Species/ strain	Variables	Number	Average daily does (mg/kg/ day)	Cancer types	Lowest observed adverse effect level
Della Porta et al. 1987	Mouse, B6C3F1	male/female oral gavage[a] (2500 or 5000 ng/ kg/wk) once per week for 52 weeks	40-50 per dose group including controls	0, 351, 714	females and males: hepatocellular (liver) adenomas and carcinomas	females: 351 ng/ kg/day for liver adenomas and carcinomas males: liver carcinomas
Kociba et al. 1978 Goodman and Sauer 1992	Rat, Sprague-Dawley	male/female oral—lifetime feeding for 2 years	50 each per dose 86 in vehicle control groups	0, 1, 10, 100	females: liver, lung, oral cavity males: adrenal, oral cavity, tongue	females: 10 ng/ kg/day for liver hyperplastic nodules in females males: 1 ng/kg/day for squamous cell carcinoma of nasal turbinates/hard palate
NTP 1982	Mouse, B6C3F1	male/female oral gavage twice per week for 104 weeks	50 each per dose 75 in each vehicle control group	0, 1.4, 7.1, or 71 for males 0, 5.7, 28.6, or 286 for females	females: lymphoma or leukemia, liver, fibrosarcoma, thyroid adenoma males: liver, lung	females: 286 ng/kg/ day for all tumours males: 71 ng/kg/day for liver tumours
NTP 1982	Rat, Osborne-Mendel	male/female oral-gavage twice per week for 104 weeks	50 each per dose 75 in each vehicle control group	0, 1.4, 7.1, or 71	females: adrenal, liver, subcutaneous tissue (fibrosarcoma) males: thyroid, adrenal, liver	females: 71 ng/kg/ day for all tumours males: 1.4 ng/kg/ day for thyroid gland follicular-cell adenoma
NTP 2006	Rat, Harlan Sprague-Dawley	female oral-gavage 5 days per week for 2 years	53 or 54	0, 2.14, 7.14, 15.7, 32.9, or 71.4	liver, lung, oral mucosa, pancreas	22 ng/kg/day for liver cholangiocarcinoma
Toth et al. 1979	Mouse, Outbred Swiss/H/Riop	male oral-gavage once per week for 1 year	43 or 44 38 in vehicle control group	0, 1, 100, or 1000	liver	100/ng/kg for unclassified hepatomas (liver cancers)

[a] *Gavage = stomach tube feeding.*

How TCDD leads to cancer is not known, but its inability to directly damage DNA or cause mutations points to indirect, non-genotoxic mechanisms, such as oxidative stress leading to production of DNA-reactive oxygen radicals. Extensive data from lab tests show TCDD can induce oxidative stress in different tissues (Stohs and Hassoun 2012). In addition, through mechanisms that may differ depending on the type of tissue, TCDD causes biochemical effects that may promote cancer development, such as elevated levels of TSH leading to thyroid cancer or changes in Vitamin A status leading to lung cancer (Schrenk and Chopra 2012). TCDD's overall effect is to promote cell division and prevent cell death, both of which can speed growth of new tumours. These mechanisms provide a biological basis for TCDD to be classified as a multisite carcinogen.

Table 3.5 lists the animal bioassays that met the screening criteria the U.S. EPA set in 2012 for use in dose-response modelling for cancer risk assessment. Although the U.S. EPA has not completed its cancer-based risk assessment, various national and international agencies have used these data for risk assessment. The Joint FAO/WHO[2] Expert Committee on Food Additives has determined that the TCDD's indirect carcinogenic effects justify the assumption of a cancer induction threshold and that a tolerable intake could be established for all adverse effects of TCDD, including cancer. The committee set a tolerable monthly intake for TCDD of 70 pg/kg/month (approximately 2.3 pg/kg/day).

3.8. The aryl hydrocarbon receptor (AhR): A unifying mechanism for TCDD's potent toxicity

TCDD induces toxicity in an unusually wide range of tissues and organs at very low concentrations. In addition, TCDD serves as a prototype for several other chemicals of environmental concern that induce a similar pattern of toxicity. These *dioxin-like chemicals* (DLCs) are structurally similar to TCDD and include chlorinated and other halogenated dibenzo-*p*-dioxins and dibenzofurans and biphenyls. Over the past 40 years, how TCDD and other DLCs cause their common pattern of toxicity has been extensively studied. The breakthrough in understanding came with the discovery of the *aryl hydrocarbon receptor* or *AhR* (reviewed in Gasiewicz and Henry 2012).

AhR—discovery of a TCDD-binding protein

When animals, including humans, are exposed to foreign chemicals (*xenobiotics*), the body responds by producing enzymes, especially in the liver. These enzymes are designed to metabolize the chemicals to a form that can be excreted. Exposure to chemicals known as *polycyclic aromatic (or aryl) hydrocarbons* (PAHs) causes the production of an enzyme known as aryl *hydrocarbon hydroxylase* (AHH; now known as *cytochrome P4501A1*).

In 1972, Nebert et al. reported that ability to induce AHH activity was an inherited genetic trait with differences among laboratory mouse strains; they called this genetic region the *Ah locus.* C57Bl/6 (B6) mice were shown to respond to the PAH known as *3-methylcholanthrene (3-MC)* by inducing AHH, while DBA/2 (D2) mice were unresponsive to 3-MC (i.e., AHH was not induced).

Around the same time, Poland and Glover (1973) were investigating TCDD as a cause of porphyria cutanea tarda (PCT), a skin disease linked to changes in production of the iron compound heme, seen in workers at plants that manufactured 2,4,5-trichlorophenol. TCDD was found to strongly *induce ALA synthetase* (an enzyme involved in heme biosynthesis) in chick embryos, implicating TCDD as a cause of PCT.

[2] FAO = Food and Agriculture Organization of the United Nations; WHO = World Health Organization.

Since TCDD is an aryl hydrocarbon, they tested its ability to induce AHH and found it was a powerful inducer of the enzyme as well. In fact, in B6 mice TCDD was 30,000 times more potent than 3-MC in inducing AHH activity. TCDD also induced AHH activity in D2 mice that were non-responsive to 3-MC, albeit with lower magnitude compared with B6 mice. These results were strong evidence that the Ah locus regulated the ability to respond to TCDD and suggested that the product of the Ah locus differed in some way in D2 mice compared with B6 mice (Poland et al. 1974). Subsequent studies showed that the product of the Ah locus was a protein that directly bound radio-labelled TCDD and that the difference between responsive and less-responsive mouse strains was due to a single codon difference in the gene that altered the protein's binding affinity for TCDD, 3-MC, and other inducing chemicals. This TCDD-binding protein was called the *aryl hydrocarbon receptor* (Poland et al. 1976).

The primary protein structure of AhR is highly conserved across a broad range of species, including humans. Human AhR is similar to the D2 mouse with a valine in the comparable position and a 10-fold lower binding affinity for TCDD compared with AhR in B6 mice (Pohnjanvirta et al. 2012). *Single nucleotide polymorphisms* (a common type of genetic variation) have also been identified in human AhR that may result in varying individual responses to TCDD (Rowlands et al. 2010).

AhR—a transcription factor

Based on the ability of TCDD to induce the synthesis of new protein (i.e., AHH), Poland et al. (1976) hypothesized that AhR acts like a steroid hormone receptor to cause specific changes in gene expression. Over the following decade, new data confirmed this basic hypothesis. However, unlike steroid hormone receptors, AhR belongs to the *Per-Arnt-Sim (PAS)* family of receptor proteins. Detailed studies of TCDD's regulation of the AHH (Cyp1a1) gene have provided convincing evidence for AhR-regulated gene transcription in many species (Ma 2012). According to this model, TCDD passively diffuses into a cell where it binds to AhR protein in the cytoplasm. When TCDD binds, other proteins dissociate from the AhR, and the TCDD-AhR complex moves into the nucleus. Another protein, AhR nuclear translocator (ARNT), interacts with the TCDD-AhR complex to form a heterodimeric transcription factor that selectively binds to a sequence of DNA known as *dioxin-response element (DRE)*. Multiple DREs are present in the upstream regulatory region of the Cyp1a1 gene. DREs are also present throughout the genome of mice, rats, humans, and other vertebrates. Inappropriate changes in gene expression in tissues throughout the body induced by TCDD-activated AhR are consistent with the diverse biological and toxicological effects observed following exposure to TCDD.

AhR-dependent TCDD toxicity

Experts agree that the first step leading to TCDD's toxicity is activation of the AhR.

Conclusive evidence for AhR-dependent toxicity came from studies of *AhR-knock-out mice*, experimental mice that have no functional AhR protein. When these mice were exposed to high TCDD doses, they did not exhibit the toxicities that AhR-expressing mice do, including acute wasting syndrome, liver toxicity, fetal malformations, or immunotoxicity. Similarly, when different types of cells from different species, including humans, were grown in a lab setting and exposed to TCDD, TCDD-related responses were blocked by interfering with AhR expression or function.

Activation of AhR by TCDD causes many changes in gene expression that influence cells' ability to regulate their growth and differentiation. Complete understanding of TCDD toxicity depends on determining specific genes that are regulated by TCDD-activated AhR and the cell-signalling pathways that are disrupted by these changes in gene expression (Swanson 2012). Determining these genes is a major undertaking as they are likely to differ by cell type, cell differentiation state, TCDD dose, and species. Connecting changes in specific genes to specific toxic endpoints will also be difficult as secondary and downstream changes in expression of other genes will contribute to the toxic response. To date, only a few specific target genes and signalling pathways have been associated with particular toxicity endpoints. Nonetheless, knowing AhR is the common primary mechanism responsible for TCDD toxicity across species is a great advancement in understanding and evaluating the potential risk of TCDD exposure in humans.

Nonetheless, knowing AhR is the common primary mechanism responsible for TCDD toxicity across species is a great advancement in understanding and evaluating the potential risk of TCDD exposure in humans.

AhR and cancer

Treating mice, rats, or hamsters with TCDD over their lifetimes caused tumours to develop in several different tissues, including liver, thyroid, lung, skin, and mouth. Since TCDD does not directly damage DNA or induce mutations and causes tumours only after long-term treatment, TCDD is considered an indirect carcinogen, promoting tumour growth by activating the AhR (Schrenk and Chopra 2012). Extensive evidence shows that TCDD activation of AhR affects the ability of cells to regulate their growth, differentiation, and programmed death, all processes known to promote tumour development. Secondary DNA damage due to induction of oxidative stress and changes in expression of cancer-causing genes are plausible mechanisms for TCDD's carcinogenicity.

TCDD—the most potent (but not the only) AhR ligand

In addition to TCDD, AhR can be bound and activated by many different polycyclic and halogenated aromatic hydrocarbon compounds (DeGroot et al. 2012). Many of these AhR-binding chemicals (*ligands)* also contaminate the environment as products of combustion, such as benzo(a)pyrene (BAP) in cigarette smoke and diesel exhaust, or from industrial use, such as some polychlorinated biphenyl (PCB) congeners and polybrominated diphenyl ethers.

Interestingly, many natural compounds in foods, particularly flavonoids and polyphenols, are also AhR ligands (Safe et al. 2012). Most of these natural compounds have AhR binding affinities that are many orders of magnitude lower than TCDD. However, they are consumed in food at levels many orders of magnitude higher than TCDD.

Possible *endogenous* (internal) AhR ligands have also been identified. Of particular interest are the photo-oxidation and degradation products of tryptophan, some of which have very high AhR binding affinity. TCDD also causes tryptophan metabolism in the liver and in immune cells by inducing tryptophan-2,3-dioxygenase and indolamine-2,3-dioxygenase, respectively. The metabolites of tryptophan—kynurenine and kynurenic acid—have been shown to activate AhR and may be the long-sought natural ligands of AhR. Arachidonic acid metabolites and modified low-density lipoproteins (LDL) are also possible AhR ligands.

For the panel's report, other AhR ligands consumed in food or generated within the body add a level of complexity when applying biomarkers of TCDD exposure, as many of these compounds can raise the base expression levels of current biomarkers such as Cyp1a1. The presence of these ligands could also raise or lower the threshold of AhR activation necessary to alter gene transcription and thus toxicity.

Regulation of TCDD as an AhR ligand

Accepting AhR as a common action mechanism for TCDD and other dioxin-like chemicals such as the PCDD/Fs and biphenyls (PCBs) led to a new approach to risk assessment based on *toxic equivalency factors* or TEFs (Tuomisto 2012). Congeners that meet the structural requirements for AhR binding (planarity and lack of ortho chlorines for PCBs) are each given a TEF value based on their potency to activate AhR and/or cause AhR-dependent toxicity. TEF values vary from 0 to 1, with TCDD = 1. *Total toxic equivalents* (TEQs) for mixtures can then be calculated based on the abundance of different congeners. The TEF method for polychlorinated dibenzo-*p*-dioxins and dibenzofurans was first proposed by the Ministry of the Environment (MOE)[3] in 1984.

This simple summation method has several shortcomings (Tuomisto 2012), including lack of consideration for interactions among congeners (especially *antagonism*, when low-affinity congeners are at much higher concentrations than high-affinity congeners), differences in persistence of individual congeners, and lack of toxicity data for some congeners. Also, individual TEFs are assigned based on laboratory animal data, and differences in ligand binding and the AhR-regulated gene set may differ between humans and rodents. In one study, a minority of AhR-regulated genes were the same between mice and humans (Flaveny et al. 2010). Nonetheless, the TEQ approach and current TEFs have been adopted internationally as the best way to estimate potential health risks of mixtures of dioxin-like AhR-acting chemicals and have served reasonably well in risk management of DLCs (Tuomisto 2012).

3. 9. Dose-response analysis of TCDD toxicity

The preceding section was a short overview of the vast array of scientific literature on the hazards (i.e., toxicity) identified in laboratory animals exposed to specific amounts of TCDD. *Hazard identification* based on laboratory animal studies is a critical part of risk assessment, which relies on identifying hazards induced at the lowest exposure levels, from which *safe* exposure levels are extrapolated.

The U.S. EPA (2012) conducted a recent, comprehensive review of these databases to identify animal bioassays for use in TCDD dose-response analysis. Based on a thorough and transparent process described in the 2012 report, the U.S. EPA identified 62 non-cancer and six cancer studies that met the criteria for dose-response modelling. Table 3.4 summarizes the reference doses for non-cancer endpoints derived from each study. The U.S. EPA has not yet reported on dose-response modelling for the cancer studies summarized in Table 3.5. However, if the lowest observed adverse effect levels (LOAEL) for cancer (thyroid tumours in male rats (1.4 ng/kg/day) and male mice (1.0 ng/kg/day)) are compared with the LOAELs for non-cancer endpoints (Table 3.4), the other endpoints are just as sensitive, e.g., reproductive effects in male rats (1.0 ng/kg/day). Since TCDD requires chronic lifetime exposure to induce cancer, dose-response modelling using cancer endpoints would likely not result in reference doses lower than those calculated for non-cancer endpoints—unless a non-threshold model was used.

[3] In most cases, the names of organizations referred to in this report have changed several times during the more than three decades covering the period of use of 2,4,5-T in Ontario. The panel chose to refer to them by their most common or current designation.

However, as previously stated, the Joint FAO/WHO Expert Committee on Food Additives (WHO JECFA 2002) concluded that TCDD's indirect mechanism of carcinogenicity justifies the assumption of a threshold for cancer induction and that a tolerable intake could be established for all adverse effects, including cancer. Health Canada (2010) and MOE (2011) have adopted the committee's tolerable daily intake for TCDD of 2.3 pg/kg/day (Table 3.6).

The World Health Organization concluded that the indirect mechanism of TCDD's carcinogenic activity justifies the assumption of a threshold for cancer induction and that a tolerable intake could be established for all adverse effects, including cancer. The World Health Organization also concluded that safe TCDD exposure levels—the so-called reference dose—would also be protective of cancer effects as well. Health Canada and Ontario's Ministry of the Environment have adopted the World Health Organization's reference dose of 2.3 pg/kg body weight/day.

Table 3.6. *Toxicological reference values (tolerable daily intake) by endpoint for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Source	Chemical of interest	Endpoint	Reference value[a]
Newton and Norris 1981	2,4,5-T	acute	20 µg/kg BW/day
US EPA 1989		chronic	10 µg/kg BW/day
US DHHS 1998	TCDD	acute	200 pg/kg BW/day
Health Canada 2010, MOE 2011, WHO 2002		chronic	2.3 pg/kg BW/day
US EPA 2012		chronic	0.7 pg/kg BW/day

[a] *BW = body weight.*

3.10. Conclusions

Epidemiological and toxicological investigations have addressed a wide range of outcomes to 2,4,5-T and TCDD exposures. Cancer was once the major focus, but studies have also looked at miscarriages and birth defects, chloracne, diabetes, heart disease, Parkinson disease and other neurological outcomes, immune system defects, and thyroid function. Reviews of the epidemiological and experimental literature have considered these outcomes. The conclusions of agencies and institutions that have performed formal reviews using panels of distinguished scientists provided the basis for the 2,4,5-T panel's hazard assessment. The panel's conclusions were as follows:

3.10.1. Chloracne

The panel accepted that TCDD is a thoroughly documented and well-accepted cause of chloracne (NRC 2006).

3.10.2. Cancer

Most agencies or institutions—including the International Agency for Research on Cancer (IARC 1997), U.S. EPA (2003), the recent IARC update (Cogliano et al. 2011), NTP (2011), U.S. EPA Science Advisory Board (US EPA SAB 2011), and the IOM (2012)—concluded that TCDD causes cancer in humans. In its review of the U.S. EPA (2003) document, the National Research Council (NRC 2006) was split over whether TCDD should be classified as *carcinogenic to humans* or *likely to be carcinogenic to humans.* Increases in all cancers combined had the strongest link with TCDD, but specific sites mentioned were non-Hodgkin lymphoma, soft tissue sarcoma, chronic

lymphocytic leukemia, and cancers of the larynx, lung, and prostate. The World Health Organization (WHO 2003) concluded that limited evidence was available for an association between chlorophenoxy herbicides in drinking water and non-Hodgkin lymphoma but that none showed an association with Hodgkin disease or soft tissue sarcoma. The American Conference of Governmental Industrial Hygienists (ACGIH 2011) said that 2,4,5-T was not classifiable as a human carcinogen but did not evaluate TCDD.

Individual reviews of cancer hazard led to different conclusions. Crump et al. (2003), Steenland et al. (2004), and Guernsey (2007) concluded that TCDD causes cancer, while Starr (2003), Cole et al. (2003), and Boffetta et al. (2011) determined it does not. Concerns and issues raised by those doubting the relationship include:

- making inappropriate assumptions about the half-life of elimination and modelling TCDD as an initiator rather than a promotor (Starr 2003)
- using the non-threshold dose-response model
- failing to consider confounding by smoking and other occupational exposures
- inappropriately considering an association with all cancers combined instead of focusing on specific sites
- moving TCDD to the known carcinogen category after decades of being listed as a suspected carcinogen (Cole et al. 2003)
- introducing possible publication bias, not considering the unusually long latency for TCDD as a promoter, and emphasizing all cancers combined rather than specific sites (Boffetta et al. 2011)

The panel accepted the largely unanimous opinion voiced in institutional and agency reviews that TCDD causes human cancer (IARC 1997, U.S. EPA 2003, NTP 2011, IOM 2012). These evaluations noted the relative consistency of the findings from most individual studies, the existence of exposure-response gradients in several studies, and the lack of fatal epidemiological weaknesses (e.g., chance, confounding, or bias) that would cast doubt on these conclusions. The panel felt the criticisms that some reviewers raised were not of sufficient likelihood or concern to discount reported associations.

3.10.3. Heart disease

Humblet et al. (2008) concluded that TCDD exposure was associated with ischemic heart disease and all cardiovascular disease mortality and that studies with better exposure assessment and use of internal, rather than external, comparison populations showed stronger associations. Exposure-response trends were observed in some studies. The IOM (2010) concluded that limited evidence was available for an association between TCDD and ischemic heart disease and hypertension. The primary limitation they noted was the lack of adjustment for potential confounders. However, NRC (2006) indicated that this link was not convincingly supported by the scientific literature.

The panel, based on the Humblet et al. (2008) review and the IOM (2010) conclusion of limited evidence, concluded that it is prudent to assume TCDD exposure and heart disease could be linked.

3.10.4. Other outcomes

A few studies have focused on the possible relationship between TCDD exposure and diabetes, neurological outcomes including Parkinson disease, miscarriages and birth defects, and immune system defects. The IOM (2010) concluded limited evidence was available for an association between TCDD and early-onset transient

peripheral neuropathy and Parkinson disease, but the NRC (2006) said the evidence did not yet show clear associations with neurological endpoints. In their review of diabetes, Remillard and Bunce (2002) concluded that the epidemiological literature showed that exposure to low TCDD concentrations was weakly correlated with diabetes and that TCDD interaction with the Ah receptor might antagonize peroxisome proliferator-activated receptor functions and promote diabetes onset. The NRC (2006) thought the evidence for an association between TCDD and diabetes was limited, while the IOM (2010) felt sufficient evidence was available to show an association.

The panel accepted these institutions' conclusion of limited evidence for an association between TCDD and Parkinson disease, peripheral neuropathy, and diabetes.

3.10.5. Mechanism of action

AhR activation is considered to be an essential primary event that underlies all toxicities associated with TCDD exposure in both humans and laboratory animals. The mechanism of AhR action is essentially the same in all species and supports extrapolating findings in laboratory animal studies to humans for risk assessment purposes. It also supports using a threshold model to assess risk for both cancer and non-cancer endpoints.

3.10.6. TCDD toxicological reference values

The panel concurred with the Joint FAO/WHO Expert Committee on Food Additives that a TCDD tolerance value of 2.3 pg/kg/day is appropriate and protective for all health endpoints, including cancer. This conclusion is consistent with Health Canada (2010), MOE (2011), and CCFME (2002).

Table 3.6 presents a summary of toxicological reference values established by various governments and agencies.

3.10.7. Uncertainty assessment

Both toxicological hazard assessment in laboratory animal studies and epidemiological assessment in human populations offer strengths and weaknesses in study design and in informing risk assessment of potentially exposed populations. Laboratory studies can limit and effectively control many variables, but extrapolating results of these studies to human populations is difficult. Conversely, results of studies in human populations can be applied directly to understanding potential adverse health outcomes in people, but controlling the wide array of variables in human populations is complex and difficult and often makes it challenging to use the results of these studies. Taken together, both toxicology and epidemiology studies can offer important insight when establishing causes of adverse health outcomes in human populations.

The panel carefully considered the strengths and uncertainties of both toxicology and epidemiology studies and the possible effects on study outcomes. Table 3.7 presents the assessment of uncertainties related to toxicology and epidemiology.

Table 3.7. *Assessment of uncertainties in toxicology and epidemiology studies related to 2,4,5-trichlophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Assumption	Justification	Effect on analysis
Exposure and outcome can be misclassified in all epidemiological studies, but effect on relative risks can be evaluated.	Study design, data characteristics, and the scientific literature provide information on the likelihood of differential or non-differential misclassification.	• Non-differential misclassification occurs in all studies and biases relative risks towards the null. • Differential misclassification may occur and can result in either over- or underestimates of relative risks. • Non-differential misclassification flattens real exposure-response relationships.

Confounding may be but is not always present and can be evaluated and controlled.	With epidemiological studies, confounding can be controlled by study design or statistical adjustment using information on supposed confounding factors.	• Uncontrolled confounding can:
		  • artificially increase or decrease relative risks
		  • result in false positive or false negative associations

The healthy worker effect can occur in cohort studies that use the general population as the referent. Studies with an internal comparison of unexposed workers are stronger methodologically.	The scientific literature has demonstrated the healthy worker effect, but it is stronger for some diseases than others and is not the same in all studies.	Relative risks from studies using the general population as the referent should be considered possibly biased towards the null.
Study power and statistical variation are important considerations for evaluating the strength of epidemiological study findings.	Epidemiological studies provide information needed to evaluate statistical variation, and the epidemiological literature provides guidelines for such assessments.	• Relative risks with wide confidence intervals may be chance findings. • Studies with low power may generate chance positive associations, as well as miss real associations.
Multiple exposures occur in all epidemiological studies. Exposure-response patterns can be used to separate effects of different exposures.	For two exposures to show the same exposure-response pattern, they must be perfectly correlated or misclassified to the extent to produce perfect correlation.	Assessing the degree of correlation between different exposures is needed to assign exposure-specific relative risks.
Results from studies with different designs may be combined in summaries and reviews.	Strengths and limitations of different epidemiological designs are well understood and can be considered in literature reviews.	Considering strengths and weaknesses of different designs provides critical information on the overall evaluation.
High-dose lab studies can represent much lower-dose human exposures.	Laboratory toxicology studies are typically conducted at very high doses—often tens of thousands of times greater than anticipated human exposures—to compensate for lack of statistical power associated with small group sizes in laboratory studies.	High dose studies may over-represent potential adverse effects at exposure levels more typical of the human experience.

Assumption	Justification	Effect on analysis
Studies in laboratory animals can reliably predict outcomes in human populations.	Due to ethical considerations, toxicology studies are typically conducted using laboratory animals rather than humans. While the ethical justification is clear, the challenge is then to relate the laboratory animal outcomes to potential adverse human outcomes, when people may actually be more or less sensitive to the toxic effects.	• Laboratory animals could over- or under-represent potential adverse outcomes in human populations. • In the case of dioxins, the scientific community widely accepts that some species of laboratory animals may be more sensitive to dioxin exposures than humans (which the laboratory animals are intended to model).
Using rigorous and transparent selection criteria will identify the most scientifically justified key studies for dose-response modelling.	A process must be established for selecting key studies from the very large TCDD toxicity database. Key issues: toxicologically relevant endpoints, relevant route of exposure, dose range inclusive of low doses, sufficient numbers of animals per group, scientifically valid study design.	This process provides the basis for risk assessment. If the best studies are not chosen, the risk assessment could under- or overestimate true risk.
Physiologically based pharmacokinetic modelling is appropriate for simulating TCDD blood concentrations in animal studies for estimating doses. Human exposure estimates can be reliably extrapolated	Most animal studies involve dosing the animals with TCDD orally or via food; the actual circulating TCDD concentration in blood is usually not measured.	Modelling has many uncertainties that could raise or lower risk assessment results.
Exposure-response data from animal studies can be used to derive the dose response at exposure levels below those used in the actual studies.	Depending on the data available, no observed adverse effect levels, lowest observed adverse effects levels, and/or benchmark dose modelling can be used to determine the critical exposure level for the toxic effect.	Modelling has many uncertainties that could raise or lower risk assessment results.
A valid reference dose can be derived from the dose-response analysis by applying safety factors.	The human population is not homogeneous in age, health status, or sensitivity to chemical toxicity. Lowering the reference dose to account for these differences is good practice.	Safety factors may be insufficient to protect or excessive.

4. Exposure assessment

4.1. Charge question

As part of its mandate, the Independent Fact-Finding Panel on Herbicide 2,4,5-T was asked to:

> *Document the methods 2,4,5-T herbicide was deployed by employees of provincial ministries and agencies or their contractors, and the interaction of those employees and the general public with 2,4,5-T herbicide application operations in affected areas* (Province of Ontario 2011).

4.2. Introduction

The objective of the exposure assessment was to predict potential human exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) using available documentation to develop the exposure scenarios and pathways identified below.

The objective of the exposure assessment was to predict potential human exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).

Estimates of exposure to the contaminants, or chemicals of concern, are based on these parameters:

- herbicide use patterns and application rates according to Ontario government records, provided in the Ministry of Natural Resources (MNR)[1] database
- occupational and non-occupational activities as defined by government operation manuals, standards, pesticide handler assumptions, and information about the nature and location of use in Ontario
- the physical/chemical characteristics of chemicals of concern that determine their interaction with and behaviour in surrounding environment (e.g., water solubility, volatility, tendency to bind to particles, etc.)
- the characteristics of the environmental compartments where spraying occurred (e.g., air, soil, plants, etc.), as well as the quantities of chemicals entering the compartments from various sources and their persistence in these compartments
- the behavioural characteristics of the human receptors that determine the actual exposures through interactions of the receptors with the various pathways (e.g., respiration rate, body weight)
- the equations and algorithms used to predict exposures to the receptors

Whenever possible, the panel selected physiological factors such as body weight, inhalation rate, and behavioural factors such as incidental soil ingestion to be consistent with Health Canada and Ministry of Environment (MOE). Occupational and bystander exposures were estimated based on historical spray information (see Section 4.3). Exposure point concentrations in various environmental media (soil, dust, wild berries), used for the recreational visitor, resident, and First Nations assessments, were predicted using the equations and assumptions in Section 4.4 and historical spray information in Section 4.3.

[1] In most cases, the names of organizations referred to in this report have changed several times during the more than three decades covering the period of use of 2,4,5-T in Ontario. The panel chose to refer to them by their most common or current designation.

The exposure assessment has provided a range of potential exposures for each scenario. The range was described by three individual estimates of exposure: low, central, and high. The high estimate was based on a series of individual worst-case assumptions, applied one after another, introducing a repetitive input parameter selection bias. The high-case estimates represent a worst-case situation, while the central and low exposure estimates are based on, whenever possible, central (average) and low-end exposure parameters, respectively. Some of the parameters that vary within the exposure estimates include TCDD content in 2,4,5-T, use of personal protective equipment, fraction of herbicide penetrating the forest canopy, and half-lives of TCDD and 2,4,5-T in soil. Not all input parameters were associated with a range (i.e., low, central, high). For example, body weight, soil ingestion, etc., were assigned a single value that was applied to each exposure estimate. A range of potential exposure values (via the use of selected low, central, and high input parameters) were developed to provide a general sense of the level of uncertainty and variability present within the quantitative exposure estimates.

To predict environmental media concentrations (e.g., soil, surface water, plants, wild berries, etc.) resulting in non-occupational exposure, the panel assumed an average annual application rate and a theoretical area. Assuming the generic application rate over a given application duration, and a steady rate of degradation within the environment, the resulting soil concentration at the end of the application duration was estimated for 2,4,5-T and TCDD. These soil concentrations were then used to determine levels in wild berries, plants, and wildlife.

The nature of exposure depended on the historical use data for each spray scenario and the information available about herbicide applicators and/or non-occupational receptors. In some cases, herbicide spraying may have occurred only once in a particular area (MNR scenario), while in others, spraying occurred annually or semi-annually. Bystanders would have been present for only a single spray event per year.

The panel's sources of information for the exposure assessment were as follows, with citations provided in text as appropriate:

- Historical spray information (herbicide used; application rates, areas, and methods; applicator characteristics; practices; etc.) was gleaned from the vast amount of historical records collected for this project and housed in the MNR database.
- Anecdotal information derived from the database and calls to the MNR call-in information phone line were used to build scenarios, define assumptions, and identify pathways.
- Scientific information was obtained from the scientific literature and was cited accordingly.

The exposure assessment included these elements:

- scenarios (the panel did not consider municipal, industrial, and private use)—forestry (MNR), Ontario Hydro, highway (Ministry of Transportation; MTO)
- chemicals—2,4,5-T and TCDD
- receptors
    - worker
    - bystander
    - resident
    - recreational visitors/hunter/angler
    - family of worker
    - First Nations people (dietary)

- pathways
  - direct dermal contact with herbicide
  - inhalation of herbicide
  - incidental ingestion of affected surface soil
  - direct dermal contact with soil
  - inhalation of re-suspended dust from soil
  - consumption of wild berries
  - consumption of wild game and fish
- population proximity—residential, remote
- duration of exposure—acute, chronic

4.3. Exposure scenarios

From 1948 to 1979, the Government of Ontario was responsible for applying herbicides throughout the province. Provincial agencies that used herbicides the most were MNR, MTO, and Ontario Hydro. Other provincial government users included Ministry of Education (MET), Niagara Parks Commission (NPC), and the Ontario Ministry of Agriculture, Foods and Rural Affairs (OMAFRA). Herbicide use by MET, NPC, and OMAFRA was relatively minor compared with that by MNR, MTO, and Ontario Hydro (e.g., OMAFRA used them for research purposes). In 1979, MOE (A0145229[2]) provided a snapshot of provincial agency use of herbicides:

From 1948 to 1979, the Government of Ontario was responsible for applying herbicides throughout the province. Provincial agencies that used herbicides the most were MNR, MTO, and Ontario Hydro.

- 1977: MNR used 43,300 kg of phenoxy herbicides, including 3,300 kg of 2,4,5-T.
- 1978: MTO used approximately 65,630 kg (active ingredients) of phenoxy herbicides, including approximately 16,200 kg of which was 2,4,5-T. Ontario Hydro used 59,115 kg of phenoxy herbicides, including 30,680 kg of 2,4,5-T. Ontario municipalities applied approximately 87,000 kg of 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-T mixtures to control weeds and unwanted brush.

Other uses of phenoxy herbicides in Ontario included those by various organizations to keep the rail lines, telephone lines, and oil and gas pipeline rights of way clear of weeds. Private use by homeowners and other companies also occurred, but estimates of the amounts for these other uses were not available. According to MOE (A0145229), these uses were small relative to those of MNR, MTO, Ontario Hydro, and municipalities.

In 1973, OMAFRA reported that 86,730 kg of phenoxy herbicides (2,4-D and 2,4,5-T in a 1:1 ratio) were applied to 51,260 ha of right of way (30 feet[3] on each side of the road) at an average rate of 1.69 kg/hectare (A0145443). Similarly, in 1978, OMAFRA reported that 74,430 kg of phenoxy herbicides were applied to 54,150 ha of right of way at an average rate of 1.37 kg/ha (A0145420/A0145442). In 1960, Ontario Hydro reported that highway departments, municipalities, railways, and utilities were the major users of herbicides in Canada (A0141556, Page 14). Ontario Hydro accounted for 27.3% of total herbicide use in Canada and approximately 80% of herbicide use in Ontario.

In this section, the panel provides a brief characterization of herbicide applications in Ontario based on the available usage data. The focus of this evaluation was three exposure scenarios for three major herbicide users:

[2] Citations in the format *A0* refer to records that are contained within the MNR searchable database. These records are available to the public, subject to Canadian copyright provisions and the Freedom of Information and Protection of Privacy Act. To access these records, visit www.ontario.ca/245T.

[3] The panel used imperial measurements when those were used in the source document.

- MNR: forestry scenario
- Ontario Hydro: transmission line scenario
- MTO: highway scenario

4.3.1. Forestry scenario

After Ontario's public forests are harvested, MNR is responsible for ensuring the regeneration of tree species with commercial value, e.g., spruce (*Picea* spp.), fir (*Abies* spp.), and jack pine (*Pinus banksiana*). However, in northern Ontario, low-value, fast-growing trees such as poplar (*Populus* spp.) often have the competitive advantage, delaying the return of the conifer forest. To help re-establish the conifer forest after harvest, in 1955 MNR set up a *conifer release* program for the 38 million ha of forest under its management. Each year, 16,000 to 20,000 ha were treated for conifer release, with chemical agents being the primary release method. When chemicals were used, no more than two treatments were generally required during the 50- to 80-year life of a particular forest, and for many forest areas only a single treatment was applied. MNR carefully assessed how effective chemical treatments were at promoting conifer growth and found them to work very well (see for example, A0169151).

To develop the exposure scenario for use of phenoxy herbicides in forests, the panel reviewed all 588 documents in which herbicide use was mentioned. Of these, 171 were forms that provided detailed data on spray operations. Other documents were reports that described in detail the procedures used, evaluation research, and other relevant information. Some of this information was redundant to information contained in the spray forms, and other information either provided data on spray activity for which no form was available or provided more data, such as specific maps and details on crews. The references were carefully compared to remove redundancies, as were appendix documents related to the same project.

Data sheets were created to extract relevant information. Each entry in the tabulated data was carefully compared to avoid counting a single operation more than once. Area treated and concentration of herbicide used was recorded in various ways; to be consistent with other studies on 2,4,5-T, the panel converted all quantities to their equivalent in gallons/acre. The panel was able to identify approximately 162,000 acres that were treated with 2,4,5-T alone or with 2,4-D (and rarely with trichloroacetic acid, TCA).

The spray maps and data tables showed that forestry spraying occurred almost entirely in remote locations and was primarily carried out using aircraft. Only about 12% of forestry locations were sprayed on the ground, via backpack sprayers or trucks. However, ground spraying could result in much greater potential exposure to 2,4,5-T. In most cases, each area was sprayed only once with 2,4,5-T. Table 4.1 summarizes data on forestry spray activities between 1952 and 1979, and Table 4.2 breaks down these data by district. Appendix 4.1 provides a more detailed summary of MNR forest spraying based on the references in the MNR database.

The spray maps and data tables showed that forestry spraying occurred almost entirely in remote locations and was primarily carried out using aircraft. Only about 12% of forestry locations were sprayed on the ground, via backpack sprayers or trucks. However, ground spraying could result in much greater potential exposure to 2,4,5-T. In most cases, each area was sprayed only once with 2,4,5-T.

A relatively small group of workers, employed by MNR and its licensees, did most of the spraying (see Section 4.5 for more details

about these workers). Of special interest was the widespread use of junior rangers to mark spray boundaries and perform other duties on the small work crews. MNR's Ontario Junior Ranger Program was designed to provide youth with a unique combination of work, learning, and life experiences. When junior rangers were involved in MNR spray programs, participants were all 18-year-old boys. The 1973 MNR aerial herbicide spray project set up and operations guide suggests using junior rangers or students as balloon markers (referred to as *balloon men*), meaning they placed brightly coloured balloons to mark the spray area boundaries and stayed in the spray zone until the operation was over (A0146700). The same document suggested that in small areas, one observer could be present with a corner balloon. Junior rangers were also assumed to have participated in backpack spraying programs. They continued to be involved in spray programs until the early to mid-1970s.

Table 4.1. *Ministry of Natural Resources spray activities—acres sprayed by year.*[a]

Year	2,4,5-T[b] only			2,4,5-T and 2,4-D[c]		
	Aerial	Ground		Aerial	Ground	
		Vehicle	Personnel		Vehicle	Personnel
1952			3			
1953	30					
1954						
1955						
1956	874			1224		
1957	5132		422			25
1958	2161					
1959	3389					
1960	1857					
1961	3860		548			
1962	1637		600			
1963	4269	137	801			
1964	4325		788			
1965	1790		196	9250	948	
1966	460		1670	4794		54
1967	942		85	6337		10
1968			1351	17,272		
1969	620		583	8682	3425	
1970			180			205
1971			833	1464		
1972			360			280
1973	296			2180		23
1974				434	65	
1975		25				
1976					30	103
1977			44	4780	44	10
1978			90	3900	90	140
1979	120			19,357		

[a] *Blank cells = data not available and/or no spraying occurred.*
[b] *2,4,5-T = 2,4,5-trichlorophenoxyacetic acid.*
[c] *2,4-D = 2,4-dichlorophenoxyacetic acid.*

4.3.1.1. MNR—aerial

From the early 1950s to the early 1960s, MNR aerial spraying (primarily fixed wing but also some helicopter) appeared to follow fairly standard procedures (A0128948, A0130761; references to specific records are cited only for illustration purposes):

- Wind speed between 4 to 6 mph (A0133185).
- Temperature less than 65 °F (A0128948, Page 12; A0133641).
- Radio communications required between the air crew and the ground crew.
- Ground control markers were initially smoke bombs and parachute markers, later evolving to helium balloons (A0128948, Page 13). Junior rangers were heavily involved as ground markers (holding and moving helium balloons), in mixing and loading operations (A0133191, A0133206, A0128598, A0133641) and flight control, records, traffic control, and water supply (A0128592).

The first helicopter spraying was in Chapleau District in 1961 using a mobile gravity-fed system for loading the herbicides (A0133134). As with most aerial spray operations, flagmen were used to guide helicopters—they carried red flags on 20-foot poles. This system was later modernized to use helium balloon markers.

By the early 1960s, MNR's procedures for aerially spraying herbicides became somewhat standardized. The following methods were used in the 1962 project and were used in future projects with minor changes:

- Helium balloons were used for markers.
- Mixing was done in three open 45-gallon drums (A0133471, A0128597)—2.5 gals of concentrate, 37.5 gals of water added per barrel. Water obtained from nearby lake using wajax pump. Spray mixture was transferred into the 120-gallon spray tank using a portable gas power pump. Nine men were used – two rangers on flight control, two rangers mixing and loading, two junior rangers mixing and recording flights, two junior rangers on traffic control, and one forester supervisor. "Junior rangers were found capable and interested and worked efficiently on the project" (A0133471).

Junior forest rangers continued to be used to mark spray area boundaries (A0131170, A0133189, A0133418), often using weather balloons as markers. The use of these relatively young workers continued during the entire period of 2,4,5-T use in Ontario: "Junior Forest Rangers proved quite satisfactory...their use on similar projects should be encouraged" (A0133189, A0131170, A0133418).

Junior forest rangers continued to be used to mark spray area boundaries, often using weather balloons as markers.

Various methods were used to transfer the chemicals to the aircraft. During early years, hand pumps were used for transfer, and chemicals were mixed by hand with wooden paddles (A0133349, A0127797, A0128686).

Health and safety considerations

The earliest references to personal protection and safety were found for 1957: "Goggles to be worn by helper on tank" (A0131233). MNR stand improvement reports from 1956-57 to 1962-63 indicated that "any man coming in contact with the brush killer should be equipped with overalls and rubber gloves for the protection of his clothes and hands since the brush killer is quite corrosive" (A0133373).

The calls for tenders for MNR's aerial brush control operations also evolved over time to include more health and safety information. The 1958 calls for tenders mentioned workmen's compensation: The "contractor has to provide satisfactory evidence that he has made suitable provisions for meeting any liabilities under the Workmen's Compensation Act of Ontario" (A0128686). By 1965, the contractor was also required to "furnish a certificate showing he is presently in good standing with the Workmen's Compensation Board of Ontario" (A0131169).

The 1958 calls for tenders also included the following working-around protection against negligence and damages: "The contractor shall use all proper precautions for the prevention of accidents and he shall indemnify and save harmless the Department from all suits and actions for damages and costs to which the Department may be put by reason of injury to persons or property resulting from negligence, carelessness or any other cause whatsoever in the performance of the work. The contractor shall be liable for all damages to persons or properties caused by reason of his operations in this contract." Wind speed was to be less than 4 mph, and temperature less than 65 °F (A0128686, A0133575). This wording was repeated in the 1962 (A0133189), 1969 (A0131325), and 1971 (A0130794) calls for tenders.

The earliest references to personal protection and safety were found for 1957: "Goggles to be worn by helper on tank" (A0131233). MNR stand improvement reports from 1956-57 to 1962-63 indicated that "any man coming in contact with the brush killer should be equipped with overalls and rubber gloves for the protection of his clothes and hands since the brush killer is quite corrosive" (A0133373).
The calls for tenders for MNR's aerial brush control operations also evolved over time to include more health and safety information.

In 1981, MNR published Aerial Spraying for Forest Management—An Operational Manual, which said that "the Contractor and the pilots must be licensed to conduct aerial spray operations in accordance with The Pesticides Act (Ontario) 1973" (A0136248).

At the very end of the period 2,4,5-T was used in Ontario, MNR began to provide health and safety comments on spray operations. In 1977 and 1978, these recommendations were provided to reduce exposures:

- "People involved with spray mixtures to be safety outfitted. ...Coveralls should be purchased for aerial spraying to eliminate people wearing their own clothing when involved with mixing" (A0128514).
- "Protective clothing to be purchased for people involved in mixing operations" (A0128514).
- "The men involved with mixing and loading were well protected with rubber gloves and boots, rubberized bib overalls and goggles" (A0133107).
- "There should be someone from MNR at the actual spray site....As he will probably get sprayed, he should be wearing a respirator and goggles" (A0133107).
- "...pleased to see the crew at the mixing site wearing hats, goggles, rain suits, rubber gloves and rubber boots....The incident on Sunday evening when about a quart of 2,4 D concentrate was dumped on one man's head proves that even the best system is not accident proof. Fortunately, due to the complete protective gear, the only result of the accident was that the worker got a bit of chemical on his hair and had to discard his goggles" (A0133107).
- "Good practice to have dry sawdust at the mixing site....For roadblocks should have a physical barrier with a stop sign on it – just having a Junior Ranger sitting beside the road was obviously not effective on Saturday evening" (A0133107).

- "Idea of using a barrel pump to transfer concentrate from 45 gallon drum to calibrated reservoir above the mixing tank is a good one. ...Should post mixing and loading site with warning signs. ... If mixing tanks are to be left unattended when full, it is desirable that inlet and outlet valves can be locked" (A0133107).
- "Goggles should be worn any time one is working with or around the chemical" (A0133107).
- "If balloon men are situated such that they are exposed to spray they should wear goggles and respirators with pesticide cartridges" (A0133107).
- "...in all cases the chemical was pumped first, followed by the water, in order that the chemical never stays for any period in the hose, which lessens the chance of accidental spills" (A0128448, A0128476, A0128496).
- "...a licensed applicator has to be (should be) present when spraying" (A0128476, A0128496, A0128505).
- "...spraying is discontinued when wind is above 5 to 7 M" (A0128505).
- "...discontinue spraying when temperature is above 65 degrees F (A0128505) or 20 degrees C" (A0128496, A0128476).
- MNR equipment included personal protective gear: rubber gloves, coveralls, and respirators (A0133107).

4.3.1.2. MNR—ground application

On selected sites, workers did ground spraying using portable or carrier-mounted mist blowers. A 1957 MNR record provides these details of a ground application: "Every man filled up his sprayer at the swamp buggy trailer with water and herbicide...walked into his area between 2 pickets (1 chain apart) directing the spray at clusters of alders and willows. Each person sprayed one strip approximately 6 feet wide and less than 3 chains long" (A0128688).

During the early 1960s, MNR was involved in a *Ribes* spp. (currant, gooseberry) eradication program to help control the spread of white pine blister rust (*Cronartium ribicola*). Workers used backpack sprayers, with usually four to five people on a crew. Junior rangers were often involved. The records do not mention safety or person protective equipment (A0128026, A0127876). One exception: "The crews systematically covered their areas by walking five abreast with the outside man laying a paper trail for a line to follow on the return run. Each man covered a distance of approximately 8' on either side of him as he progressed. Each plant was to be sprayed to the dripping-off point" (A0128027). During a 1969 *Ribes* eradication, a summer student was hired as foreman for the job: "He in turn hired an Indian Crew from the Sabaskong Reserve" (A0130607). The project continued later in the summer with junior rangers.

Table 4.2 and Appendix 4.1 provide details about where MNR spraying occurred. Given the remote locations, it is possible that only hunting/fishing and recreational areas were affected. However, it is also possible that the spraying affected First Nations lands and/or hunting/fishing areas. A generic river/lake used for fish/game and surface water activities by northern residents and visitors, including First Nations people, were considered in the exposure evaluation of MNR spray activities. At least one spray project was carried out cooperatively on First Nations land (Christian Island) to create merchantable timber (A0130768, A0130853). A small number of spray projects used First Nations crews.

Because many reports included specific maps or geographic landmarks of the areas sprayed, the panel was able to identify the locations using Google Earth©, vectorize the maps using R2V© software (Able Software 2012), and create a set of maps of the forestry spray areas. For the few reports that had no spray area maps but did have complete descriptions of the area and acreage sprayed, the panel created a placeholder polygon of the correct size, as shown in figures 4.1 and 4.2.

Table 4.2. *Ministry of Natural Resources spray activities—acres sprayed by district.*[a]

District	2,4,5-T[b] only			2,4,5-T and 2,4-D[c]		
	Aerial	Ground		Aerial	Ground	
		Vehicle	Personnel		Vehicle	Personnel
Black Sturgeon						
Blind River						
Boucher Lake						
Brockville						
Cardiff						
Chapleau	1985			1000		
Cochrane	1200			1769	25	25
Espanola					150	150
Fort Frances	296	387	137	3613		
Geraldton	2200			11,303	10	10
Gillies		234				
Gogama	4420					
Gore						
Griffith						
Haldimande and Cramahe						
Hastings						
Hearst				3508		
Huronia						
Kapuskasing	11,065			41,492		
Kemptville	44	371				
Kenora				634		
Kirkland Lake				650		
Lake Huron	403	163				
Leeds County						
Longlac	3967	551		564		
Lyndoch						
Maria						
Marten River		252				
North Bay	3444	2638		274	54	54
Ottawa	120				23	23
Parry Sound						
Pembroke	620	560	25			
Port Arthur				1026		
Red Lake				480		
Sheppard						
Sudbury					103	103
Swastika	1605					
Temagami and Gillies		500				
Temagami		374				
Thunder Bay		134		9667		
Tweed	600	1790		623	485	485
Wawa						
White River				2021		
Whitney						

[a] *Blank cells = data not available and/or no spraying occurred.*
[b] *2,4,5-T = 2,4,5-trichlorophenoxyacetic acid.*
[c] *2,4-D = 2,4-dichlorophenoxyacetic acid.*

Examination of the data in Appendix 4.1 showed that the herbicide concentration used varied over the years and from project to project. Most acreage appeared to have been sprayed at a rate of 1.5 lbs (acid equivalent) of 2,4,5-T per acre. For this exposure assessment, the panel used this value but built scenarios that provide for both higher and lower concentrations.

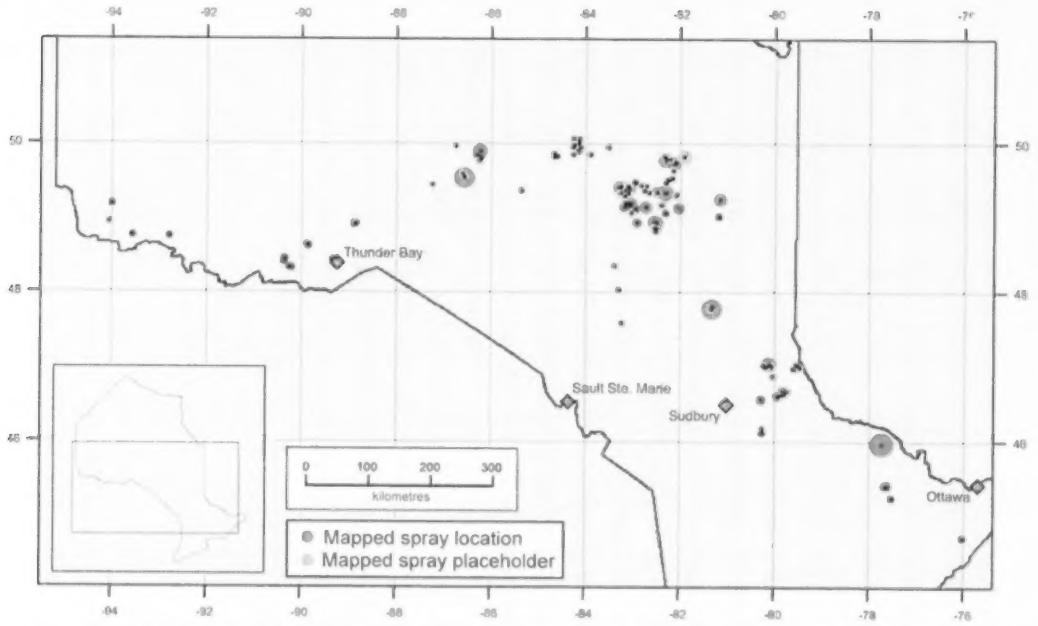

Figure 4.1. *Areas sprayed from 1956 to 1979 by the Ministry of Natural Resources (MNR), based on data from the MNR database. This map represents about 76% of the total known sprayed area (128,700 acres out of 169,425 documented as sprayed). The diameter of each coloured area represents the number of acres sprayed at a given location. The 70 areas in purple were derived from maps in the MNR database and represent 61,046 acres of 169,425 acres (36%) known to have been sprayed. The 57 areas shown in orange total 67,654 acres or another 40%; these orange areas accurately represent acreage sprayed, but their locations are approximate due to the lack of precise geographic data.*

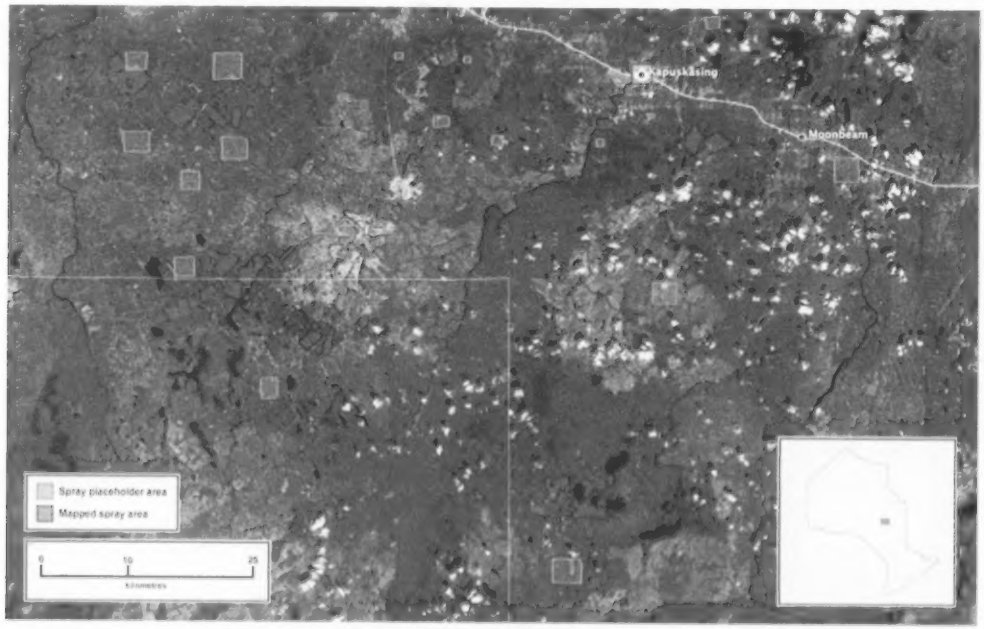

Figure 4.2. *Areas sprayed by the Ministry of Natural Resources from 1960 to 1979 near Kapuskasing. This map shows about 7.6% of the area displayed in Figure 4.1 as an example of the type of area sprayed (derived from Google Earth©). The markers represent 53,173 acres out of 169,425 total acreage (or 31.4%) for which documentation is available. About 48% of the sprayed acres (in purple) are the actual areas sprayed, as derived from the records. The remaining areas (in orange) are other acres sprayed, but their location and shape are approximate due to the lack of precise geographic data.*

4.3.2. Ontario Hydro scenario

Ontario Hydro, a Crown corporation, was responsible for generating and transmitting electrical power throughout Ontario from 1906 until 1999, when it was split into five successor companies. As part of its activities, Ontario Hydro was responsible for maintaining all high-voltage hydro transmission lines and corridors (rights of way) in the province. To ensure the safe, reliable, and economical operation of the power system, maintaining these lines requires the control (trimming, cutting removing, preventing from growing) of tall woody plants that grow along these transmission lines (A0138471).

Ontario Hydro has used herbicides to manage vegetation since 1948, when 2,4-D, 2,4,5-T, and Amate were first sprayed on 66 acres for right-of-way cleaning (A0138513; A0141556, Page 13). Spraying was primarily conducted via truck- and trailer-mounted spray units, with right-of-way width averaging 100 feet. Through the 1940s and '50s, herbicide use steadily increased with the first aerial spraying in 1952. In 1954, 14,974 acres were sprayed, including 1399 by aircraft (150 acres were sprayed experimentally by helicopter).

By 1960, more than 40,000 acres were sprayed annually, with approximately 10% by aircraft—fixed and rotary wing (A0138513). As of 1979, Ontario Hydro's Forestry Department was responsible for managing more than 40,000 km of main transmission lines and 90,000 km of rural distribution lines (upwards of 300,000 ha). Brush control was necessary on approximately 121,000 ha (300,000 acres) to control fast-growing vegetation such as maple (*Acer* spp.), ash (*Fraxinus* spp.), birch (*Betula* spp.), and poplar (A0145229). Vegetation control programs included selective cutting and herbicide spraying, typically over a three-year time frame for new lines, followed by spray intervals of six to seven years. Herbicides used (either individually or in mixtures) were 2,4-D, 2,4,5-T, Tordon 101, Tordon 10K, and fenoprop.

According to Ontario Hydro (A0145229), training for pesticide applicators was provided as part of a three-year training program. Training programs have been in place since 1949 (A0141556). One licensed person was in charge of every crew, and all staff were required to follow all safety rules regarding protective clothing and the handling and use of pesticides (A0133096). A comprehensive application manual has been available for all staff dating back to at least 1954 (A0138473). In addition to providing detailed information about spray procedures and methods, the manual provided several precautions for worker health and safety, application restriction, unfavourable weather conditions (wind, rain, heat), and disposal. For example, one document instructed: "Spray crews are required to wear long sleeve shirts, long trousers and leather gloves....When mixing or loading concentrated herbicides rubber or neoprene gloves must be worn." Safety procedures did not include the use of respiratory devices (A0148468). A 1957 Hydro Electric Power Commission contract for aerial spraying of rights of way (A0138400) specified the rate of spray per acre and warned about the need to control wind conditions to avoid drift.

In July 1979, OPAC observed an Ontario Hydro spray operation (A0148478) and wrote:

- *Three men applying the spray as a wash over the vegetation and in the process had become soaked with the herbicide solution – "extreme exposure".*
- *They wore hard hats, leather gloves and rubber boots with their pants tucked into their boots*
- *No protection of the face or for breathing*
- *As the men sprayed, they often directed the spray at each other or upwards*
- *They sprayed ahead of themselves, into the direction of travel and into the wind*
- *The work was fast paced and strenuous. Most of the breathing would have been by mouth.*

- *The three spray applicators (summer students) advised that rubber gloves are too hot and that some applicators wore no gloves at all. The leather gloves became stiff after about two weeks, when they would be discarded for new ones.*
- *Six crew members. The first three were all licensed applicators:*
  Driver of water tank to supply spray rig
  Driver of spray rig (sits in enclosed cabin)
  Individual walking ahead to direct the spray rig
- *Three applicators – summer students – most heavily exposed.*
- *At the end of the day, workers returned to their homes in their work clothes. Clothes were washed when the spray applicators felt "it was necessary."*

In 1979, MOL responded to these OPAC observations as follows: "Summer program: spray is directed upwards to foliage, men wear leather gloves because neoprene is too hot, also too hot for masks, hose handlers and gloves get soaked. In the Fall, spray is directed to base of brush and because the weather is cooler, men wear rain suits, masks, neoprene and hard hats" (A0145430).

A study published by the Ontario Hydro Employees Union in 1984, after all use of 2,4,5-T had ended, may provide further insight into how the herbicide had actually been used, although this is not certain: The "majority of foresters" used gloves, coveralls, goggles, etc., while spraying, as advised by management, and fewer (but some) said that they had been advised to do so by the union. However, only 20% said they showered before leaving work, fewer than 25% claimed to wear a respirator while spraying, almost 50% wore work clothes after work, 90% said they got herbicide on their hands and arms while spraying, and almost 50% claimed they sprayed in winds over 20 mph (A0138407, Page 4).

Consistent herbicide spray information is lacking; see Table 4.3 for a summary of known Ontario Hydro spray activities.

Table 4.3. *Ontario Hydro spray activities from 1948 to 1966.*[a]

Year	Ontario Hydro right-of-way activities			
	Total acres sprayed	Total hectares sprayed	Aerial spray	Miles sprayed (assuming 100 feet (30 m) right of way)
1948	66	27		6
1949	247	100		21
1950				
1951	3899	1578		325
1952	7971	3226		664
1953	13,608	5507	634 acres by aircraft	1134
1954	14,974	6060	1399 acres by aircraft and 150 acres by helicopter	1248
1955	20,131	8147	299 acres by aircraft	1678
1956	20,039	8109	924 acres by aircraft	1670
1957	37,046	14,992	5100 acres by aircraft	3087
1958	40,397	16,348	5778 acres by aircraft	3366
1959	37,859	15,321	4900 acres by helicopter	3155
1960	42,850	17,341	6271 acres from the air	3571
1961	45,529	18,425	5148 acres from the air	3794
1962	41,939	16,972	4176 acres by air	3495
1963	44,421	17,977	3339 acres by aircraft	3702
1964	58,710	23,759	3683 acres from the air	4893
1965	46,440	18,794		3870
1966	42,920	17,369		3577

[a] *Blank cells = data not available and/or no spraying occurred.*

Table 4.4 shows data on the volume of herbicide that Ontario Hydro purchased for the years 1973 through 1978. For these years, 2,4,5-T-containing products (2,4,5-T alone and premix) averaged 30% of total herbicides used by Ontario Hydro (A0138492), with 2,4,5-T representing 20% of the total active ingredient Ontario Hydro purchased. In earlier years, it appears that the primary herbicides used were 2,4,5-T/2,4-D combinations, and 2,4,5-T alone (A0138513).

Table 4.4. *Ontario Hydro 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and premix orders/use from 1973 to 1979.*

Year	2,4,5-T (L)	Premix (L)[a]	2,4,5-T sprayed (gals)[b]
1973	37,160	14,397	9759
1974	13,638	1568	3000
1975	25,912	12,888	5701
1976	19,116	7319	4206
1977	22,661	10,456	4985
1978	35,709	16,388	7856
1979	none	none	none

[a] *Premix = 50/50 mix of 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-T.*
[b] *2,4,5-T applied = 2,4,5-T alone + 50% of premix; 4.5 L/gallon.*

Right-of-way spraying occurred on a five- to six-year cycle, resulting in an approximation that annual spraying involved 20% of the total distance of high-voltage transmission lines each year. The total Ontario Hydro right-of-way area was reported to be 200,000 acres in 1966 and 300,000 acres in 1978. Other years were approximated based on these acreages. The distance sprayed (miles of high-voltage transmission lines) was approximated based on 12 acres/mile for the standard high-voltage right-of-way width of 100 feet. On a weight basis (assumed 1 L of herbicide is equivalent to 1 kg of herbicide), approximately 20% of the herbicides used between 1973 and 1978 contained 2,4,5-T; this breakdown was assumed for other years. Table 4.5 shows the assumed spray areas, distances, and volumes for Ontario Hydro.

Table 4.5. Assumed 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) spray areas, distances, and volumes for Ontario Hydro from 1948 to 1979.[a]

Year	Spray area (acres)	Spray distance (miles)	2,4,5-T spray volume (gals)
1948	66	6	7[b]
1949	247	21	24
1950			
1951	3899	325	385
1952	7971	664	786
1953	13,608	1134	1342
1954	14,974	1248	1477
1955	20,131	1678	1986
1956	20,039	1670	1976
1957	37,046	3087	3654
1958	40,397	3366	3984
1959	37,859	3155	3734
1960	42,850	3571	4226
1961	45,529	3794	4491
1962	41,939	3495	4136
1963	44,421	3702	4381
1964	58,710	4893	5791
1965	46,440	3870	4580
1966	42,920	3577	4241
1967	60,000	5000	5918
1968	60,000	5000	5918
1969	60,000	5000	5918
1970	60,000	5000	5918
1971	60,000	5000	5918
1972	60,000	5000	5918
1973	60,000	5000	9759
1974	60,000	5000	3000
1975	60,000	5000	5701
1976	60,000	5000	4206
1977	60,000	5000	4985
1978	60,000	5000	7856

[a] *Blank cells = data not available and/or no spraying occurred.*
[b] *Coloured font = estimated value.*

Based on information provided in A0138404, Ontario Hydro use of 2,4,5-T typically involved applying it in combination with 2,4-D at a rate of 3.5 lbs of 2,4-D and 3.5 lbs of 2,4,5-T per gallon of product, mixed at a rate of 0.5 gals of herbicide in 100 gals of water. The application rate was typically 100 gals of mix or 0.5 gals of herbicide per acre or 0.25 gals of 2,4,5-T. The application rate of active ingredient per acre was reported as 3.5 lbs a.i. (active ingredient). At 12 acres per right-of-way mile, the application rate was 42 lbs total a.i. per mile or 21 lbs of active 2,4,5-T per mile when mix was applied. Comparing Ontario Hydro use of 2,4-D/2,4,5-T premix to Agent Orange (50/50 mix of 2,4-D and 2,4,5-T) use in Vietnam indicates that the latter rates were 9.4 times greater (A0138404). Empirical calculations based on the available information substantiate the premix application rates (A0138404) and indicate that 2,4,5-T, when used alone, was applied at 3.5 lbs per acre or 21 lbs per mile.

Figure 4.3 shows the Ontario-Hydro transmission lines that were sprayed.

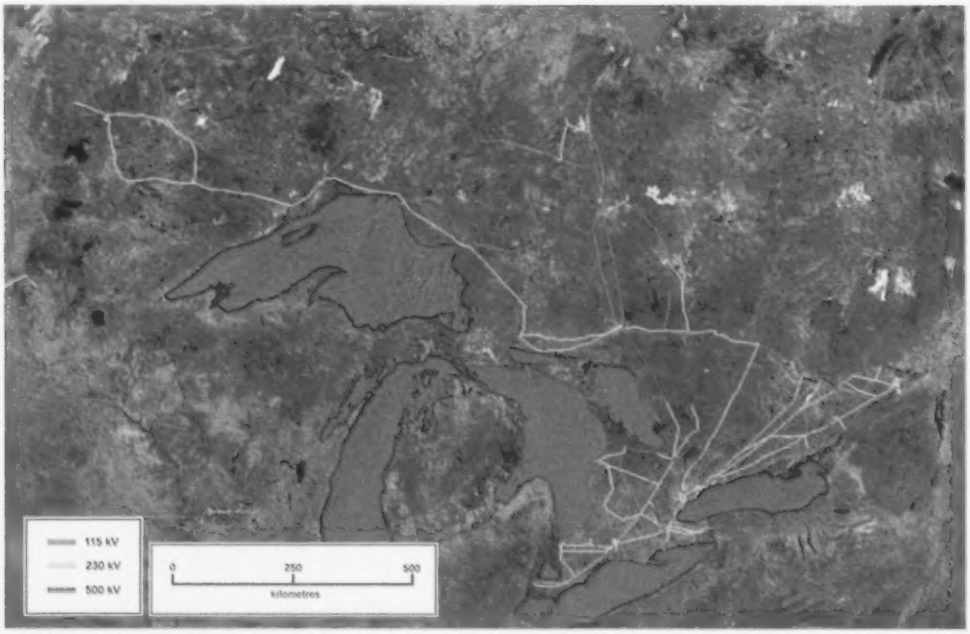

Figure 4.3. *Ontario Hydro transmission lines in 2010 (derived from Ontario Hydro's A-06-01 transmission system maps). The panel did not have access to information on changes to the transmission lines between 1952 and 2010 so this map could owerestimate areas sprayed with 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). The line width represents a 50-foot swathe on each side of the mapped transmission line (total 100-foot swathe).*

4.3.3. Highway scenario

MTO conducts weed control programs across Ontario to control noxious weeds and brush along highways. Herbicides used include 2,4,5-T alone and in mixtures with 2,4-D. A0145229 indicated that in 1979, the overall MTO spray program consisted of treating 20,000 to 30,000 ha annually (about 20% of provincial highways). This information seems inconsistent with that provided elsewhere indicating a greater annual percentage and acreage of highway right-of-way spraying. For example, in 1973, OMAFRA reported that 86,730 kg of phenoxy herbicides (2,4-D and 2,4,5-T in a 1:1 ratio) were applied to 51,260 ha of roadside right of way (30 feet on each side of the road) at an average rate of 1.69 kg/ha (A0145443). Similarly, OMAFRA reported that 74,430 kg of phenoxy herbicides (2,4-D only) were applied to 54,150 ha of roadside right of way at an average rate of 1.37 kg/ha in 1978 (A0145420, A0145442).

Furthermore, MTO Quality Standard M-300 for vegetation control provided these directives on spray frequency:

- *Medians – two year cycle*
- *Shoulder Areas*
  - *Expressway – two year cycle*
  - *Undivided highway – annual*
  - *Interchange – every other year* (A0134566)

MTO considered roadside spraying to be a skilled operation and required each applicator to be well trained and licenced under The Pesticides Act. MTO provided a roadside vegetation management manual (dating back to at least 1965; A0134563) in addition to training and supervision for all staff. Also provided were protective clothing

and equipment for the operators. Guidance was also provided for spraying in crop areas, urban areas, rest areas, and picnic sites and along guardrails and drainage ditches. A0136754 indicated that herbicide should not be applied within 100 feet of any water body that could be used for drinking (including lakes, rivers, streams, ditches, and wet areas that could be sources of potable water) or within 100 feet of built-up areas, single-family dwellings, or recreational areas.

The MTO Pesticides Spray Manual for 1976 indicated that roadside weed and brush spraying should take place at a vehicle speed of between three and six miles per hour and that during a typical work day (six to eight hours), a single MTO spray team could cover 18 to 48 miles (A0142281).

Spray activities were well documented with detailed information provided in these documents:

- annual reports—provincial
- annual weed spray reports—practices district by district
- annual district reports—spray practices for a particular district
- spray forms documenting daily spray activities

4.3.3.1. Ministry of Transportation—annual reports

These annual reports provide details on various highway-related activities (construction, repaving, bridge work) including forestry and landscaping activities. Herbicide use was mentioned often (i.e., miles and/or acres of highway/right of way sprayed), but limited information was provided about the products used and applications rates/amounts.

The panel reviewed reports for 1950 to 1986 (with the exception of 1958). Although of limited use, these reports do give an idea of how many miles of provincial highways were maintained each year, and for some years, how many were sprayed with herbicides. These reports indicate that 50-70% of provincial roads were sprayed per year from 1953 to 1962, and between 1963 and 1973, more than 90% of them were sprayed annually (Table 4.6).

Table 4.6. Annual spray activities for Ontario highways from 1953 to 1973.[a]

Year	Primary highways (miles)	Secondary highways (miles)	Tertiary roadways (miles)	Total provincial roads (miles)	Herbicide spraying (miles)
1953	7871	2710		10,580	7331
1954	7905	2606		10,512	
1955	8076	2683		10,758	
1956	8522	2395		10,917	7475
1957	8691	2362		11,053	
1958	8770	2359		11,129	5669
1959	8944	2476		11,420	6217
1960	9003	2562		11,564	
1961	9318	2547		11,865	7169
1962	9437	2673		12,110	8193
1963	9447	2763	21	12,232	10,705
1964	9749	2859	119	12,728	11,421
1965	9722	2912	123	12,758	10,135
1966	9867	2945	145	12,957	11,500
1967	9897	2977	213	13,087	14,665
1968	9964	2978	213	13,154	14,196
1969	9949	3003	220	13,172	14,191
1970	9896	2995	216	13,107	15,259
1971	9772	2994	219	12,985	
1972	9802	2979	218	12,998	14,489
1973	9774	2918	218	12,909	14,489

[a] *Blank cells = data not available and/or no spraying occurred.*

4.3.3.2. Annual weed spray reports—practices by district

These reports provided annual summaries of herbicide use by MTO district (MTO divides its responsibilities into approximately 20 of these districts; see Table 4.7). Details provided include miles of provincial highway sprayed and gallons of herbicide used in each district. Annual weed spray reports were available for 1956, 1962, 1964-1970, and 1973 (Table 4.8).

Table 4.7. Ministry of Transportation districts (Ontario).

1 Chatham	7 Port Hope	14 New Liskeard
2 London	8 Kingston	16 Cochrane
3 Stratford	9 Ottawa	17 Sudbury
4 Hamilton	10 Bancroft	18 Sault Ste. Marie
5 Owen Sound	11 Huntsville	19 Fort William/ Thunder Bay
6 Toronto	13 North Bay	20 Kenora

Table 4.8. Ministry of Transportation annual spray totals (Ontario).[a]

	1956	1962	1964	1965	1966	1967	1968	1969	1970	1973
Miles sprayed	7475	8194	11,422	10,136	11,584	14,655	14,196	14,191	15,259	
Acres sprayed										53,464
2,4-D[b] amine		3451	5382	4360	4610	6662	6558	5347	7192	11,082
2,4-D ester	4367	3245	1603	728	1549	1162	1312	858	583	0
Amine	2858									
Pre-mix			5885	9822	10,143	10,276	9129	11,421	11,643	7117
2,4,5-T[c]	1532	3109	1640	720	424	438	525	721	783	0
Dycleer										75
Tordon						658		1710	4605	10,058
Total gallons sprayed	8755	9135	14,510	15,629	16,725	19,196	17,524	20,057	24,806	28,332

[a] *Blank cells = data not available and/or no spraying occurred.*
[b] *2,4-D = 2,4-dichlorophenoxyacetic acid.*
[c] *2,4,5-T = 2,4,5-trichlorophenoxyacetic acid.*

4.3.3.3. Annual district reports—spray practices for a particular district

These reports provided annual summaries of the distance of each highway within a given district that was sprayed annually and which herbicides were used. Reports of this type are available for only these districts and years:

- District 6—Toronto: 1965-1970
- District 11—Huntsville: 1958, 1959, 1963-1973
- District 14—New Liskeard: 1967-1971

These reports are of limited use but serve as a cross-check on individual spray records and other summary reports.

4.3.3.4. Spray forms documenting daily spray activities—summary reports of spray

The MNR database includes more than 850 spray forms that detail daily spray activities and conditions. This information was not available for all districts but was provided for several districts over many years:

- Toronto: 1965-1969
- Huntsville: 1958-1959, 1961-1975
- Hamilton: 1974
- New Liskeard: 1958, 1962-1972, 1975
- Cochrane: 1958, 1960, 1962, 1965-1970, 1975-1980

These daily reports provide this information:

- district
- date of spray operation
- highway sprayed
- specific location
- gallons of herbicide used
- miles/acres sprayed
- width of sprayed area
- type of vegetation sprayed
- weather conditions
- equipment used
- vehicle and sprayer operators

4.3.3.5. Summary of MTO spray activities

Examining the available MTO summary and detailed spray information in the MNR database revealed that:

- Most provincial highways were sprayed since the 1950s.
- Incomplete records do not allow for a complete and definitive tabulation of all MTO spray activities, but information was available to allow for general assumptions and conclusions.
- Most spraying was conducted by boom trucks along the shoulders and rights of way of the provincial highways with vehicles travelling between 18 and 48 miles per day.
- Some spot spraying by hand, using backpack spray units, was conducted along roadways, especially around cloverleaf interchanges.
- Spray operations were conducted between May and September/October of each year.
- MTO used 2,4,5-T alone, 2,4,5-T and 2,4-D (amine and/or ester) mixtures, and premix extensively (on average, 46% of all herbicide use contained 2,4,5-T as the active ingredient).
- Water was the main solvent; rarely, diesel was used.
- Spraying was from road to 20- to 50-foot setback (varies somewhat and only reported in some years); on average, rights of way were assumed to be 30 feet on each side of the roadways, which represents 3.63 acres per linear mile sprayed.

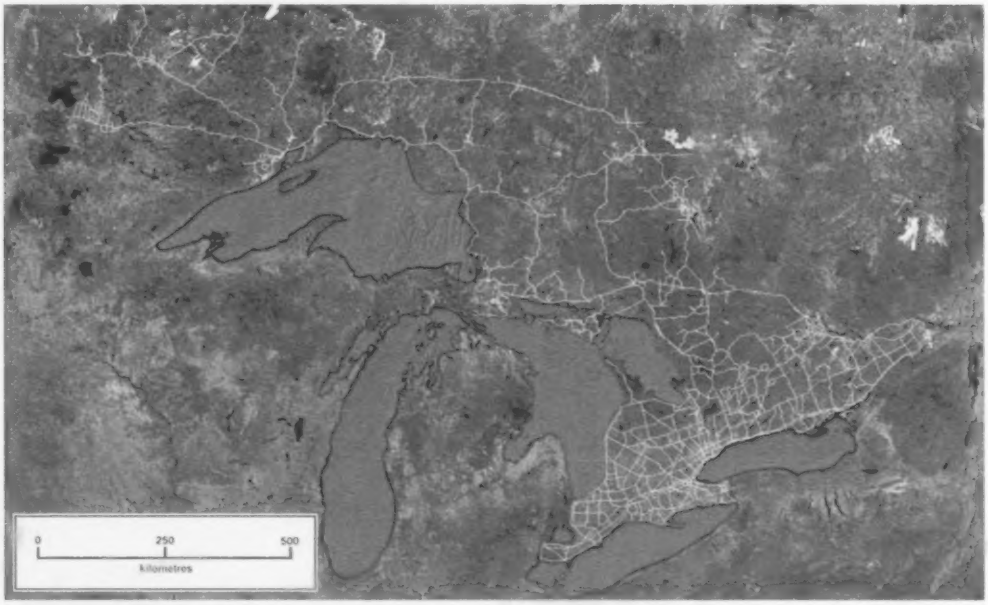

Figure 4.4. *Primary, secondary, and tertiary highways in Ontario (derived from the 1978-1979 Ministry of Transportation official road map). These roads were maintained by the Ministry of Transportation, and records in the Ministry of Natural Resources database indicate that these roads were treated with herbicides annually or semi-annually. The line widths represent a 30-foot swathe on each side of the mapped highway.*

- As per the standard protocol, spraying was stopped on windy and rainy days (seemed more diligent in some districts).
- The number of people involved in the spraying was relatively small; where documentation was available, it appeared to show that each district employed two teams of two herbicide applicators, with many workers continuing in the same role year after year (two-man teams; consistent throughout the year and year to year; two teams per district; 18 districts; maximum 72 workers per year [driver/sprayer]).
- In the peak spray years (1964-69), between 50 and 60% of the herbicide used contained 2,4,5-T, with 50 to 70% of provincial roads sprayed per year from 1953 to 1962 and 90%+ sprayed between 1963 and 1973.
- Figure 4.4 provides the panel`s best estimate of the extent of highway spraying in Ontario.

The spray records do not show a consistent application rate, which complicates estimating exposure. Interpreting the data was further complicated by the fact that the herbicide mixtures MTO used varied by district and by year. A suite of herbicides were used in varying combinations and permutations, including 2,4,5-T alone, 2,4,5-T in combination with 2,4-D, 2,4,5-T in premix formulations, as well as 2,4-D (amine and ester), Tordon, Dycleer, Karmex (diuron), and various soil sterilants. Several MTO guidance documents provide direction on formulation concentrations and application rates. For example, A0142281 indicates 2,4,5-T was used in combination with 2,4-D at rates of 0.15 to 0.3 gals per acre for most applications (this document was dated after 2,4,5-T use by MTO ceased; thus, the panel assumed it provided historical information). Average provincial application rates ranged from 1.37 kg/ha in 1978 to 1.69 kg/ha in 1973 for phenoxy herbicides (A0145420, A0145443). MTO ceased using 2,4,5-T alone in 1973, and 1973 application rates are those for 2,4-D and 2,4,5-T combined (1:1 ratio). The average application rate from 1973 of 1.69 kg/ha (approximated as 1.5 lbs/acre) was used to estimate MTO exposures.

4.3.4. Summary of personal protective equipment used

Ontario government agencies began to provide workers involved in herbicide spray operations with guidelines for using personal protective equipment during the 1950s (Table 4.9). The main concerns voiced in these early guidelines involved warnings to operators related to slipping and tripping due to oily surfaces or hoses or being burned or injured due to oil fires, hot exhaust pipes, or lifting of heavy containers. The guidelines also contained cautions related to drift, crop damage, and application during windy conditions.

Ontario government agencies began to provide workers involved in herbicide spray operations with guidelines for using personal protective equipment during the 1950s.

4.3.5. Storing, disposing of, and transporting herbicides

Information on storing, disposing of, and transporting herbicides was available in the MNR database for the time period preceding the mid- to late 1970s. In July 1970, MTO indicated that the Cochrane stores yard would be the burial ground for empty containers (A0141403), but no information was available on whether the site was used and if so, how many containers were stored there or could remain. As of 1973, storage containers may have been sent for reconditioning. The MTO recommended that the reconditioning contractor be made aware that the drums had contained a hormone herbicide: "This will enable the reconditioner to thoroughly clean the container and dispose of the residue. It is essential that the containers be thoroughly drained of all herbicides prior to recycling through the drum reconditioner" (A0141409).

Table 4.9. *Personal protective equipment suggested for Ontario government herbicide spraying field operations, including preparation, loading, mixing, and application.*[a]

Ministry/agency	1940s	1950s	1960s	1970s

Ontario Hydro	1948: first chemical brush control program			• Mixing and loading
				  • Rubber or neoprene gloves
				• Spray crew
				  • Hard hats
				  • Leather gloves
				  • Rubber boots with pants tucked in
				  • No masks

Ministry of Transportation		Chemical brush control program initiated		• Protective equipment available to all operators • "Because of equipment design there is limited exposure to undiluted chemical"

Ministry of Natural Resources		Chemical brush control program initiated
		1957: "goggles should be worn by helper on tank"
		1956-57: "any man coming in contact with the brush killer should be equipped with overalls and rubber gloves"
				• Mixing and loading
				  • Rubber gloves
				  • Boots
				  • Rubberized bib overalls
				  • Goggles
				  • Hats
				  • Rain suits
				• Balloon men
				  • Goggles
				  • Respirators with pesticide cartridges

[a] *Blank cells = information not available.*

In 1977, MNR provided these instructions: "Chemical containers should be rinsed with water and the washings added to the mixing tank. The practice of puncturing the bottom of containers right after the chemical has been emptied out results in contamination to the site and the truck used to transport the empty containers to the dump" (A0133107).

After the Ontario government discontinued using 2,4,5-T, a small inventory was left over. Ontario Hydro sold much of its remaining stock, while MNR sold its stock to the Province of New Brunswick (A0131028). Ontario Hydro continued to have about 6000 gals in stock at its Kipling facility, some of it in damaged containers (A0138500), and no records in the MNR database showed its ultimate fate. By 1980, MTO had only 295 gals left, the fate of which is unknown (A0141751). Thus, only a relatively small amount of the Ontario government's herbicide remains unaccounted for.

There was no evidence that improper storage and/or disposal of 2,4,5-T has led to potential exposures. Due to 2,4,5-T's properties, its presence in water would be the best indicator of inappropriate storage and/or disposal, but MOE data for surface, ground, and drinking water show no elevated levels of the herbicide anywhere in Ontario. TCDD—the common contaminant in 2,4,5-T—is common in the environment so its presence is not a useful marker of products containing 2,4,5-T. Thus, the panel concluded that while it cannot determine the ultimate fate of buried 2,4,5-T, no water contamination of concern has been found.

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For example, 238 stream water samples were analyzed for acid herbicide concentrations. The samples were collected in the summers of 2008, 2009, and 2010 from 10 urban streams across Ontario. Concentrations of 2,4,5-T were measured using the analytical method for acid herbicides, with the main analytes of interest being common lawn care herbicides, including 2,4-D, dicamba, and methylchlorophenoxyproprionic acid (MCPP). Researchers found 2,4,5-T in 58% (137/238) of samples, with 94% (129/137) of these >1 ng/L. The maximum concentration was 11 ng/L. MOE considered these concentrations to be very low.

Between 1974 and 1996, 2435 stream water samples were analyzed for 2,4,5-T. Samples were collected at 55 locations in Ontario as part of the Provincial Water Quality Monitoring Network. Most locations were monitored for five years or fewer, and 2,4,5-T was not detected in most samples. The few samples that showed 2,4,5-T had levels less than 1.5 µg/L, with one case where the concentration was 5 µg/L in 1975.

Between 1986 and 2006, MOE (2010) also tested for 2,4,5-T in treated drinking water samples. The herbicide was not detected in any samples collected between 2001 and 2006 (627 from ground water sources and 1,153 from surface water sources; detection limit of 50 ng/L). Not a single sample collected as part of this program exceeded the Ontario drinking water quality standard (0.28 mg/L) for 2,4,5-T. Other sampling initiatives such as Drinking Water Surveillance Program (DWSP), Drinking Water Inspections Program (DWIP), Provincial Groundwater Monitoring Network (PGMN) and Drinking Water System Regulation (O Reg. 170/03) had similar results.

4.3.6. Historical spray areas of concern

Chemical brush control with 2,4,5-T began in Ontario in 1948 and continued until 1979. The three agencies most heavily involved in chemical brush control programs were MNR, MTO, and Ontario Hydro. Ontario Hydro had the most active program and used the most product, followed by MTO and then MNR. Despite the fact that on average, MNR used only about 13% of the 2,4,5-T that Ontario Hydro used annually, and 41% of the amount MTO used (estimates taken from A0133096), more information was available about MNR's operations. Ontario government records storage and retention procedures did not require that these records be maintained, so it was fortuitous that so much information on MNR daily activities was still available. For illustrative purposes, figures 4.1 to 4.4 show where government agencies sprayed 2,4,5-T in Ontario.

Chemical brush control with 2,4,5-T began in Ontario in 1948 and continued until 1979. The three agencies most heavily involved in chemical brush control programs were MNR, MTO, and Ontario Hydro. Ontario Hydro had the most active program and used the most product, followed by MTO and then MNR.

4.3.7. Summary of spray activities in Ontario

Table 4.10 summarizes Ontario government agencies' 2,4,5-T spray activities.

Table 4.10. *Ontario government's annual 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) spray activity from 1948 to 1979.*[a]

Year	Distance/area sprayed			
	MTO[b] (miles)	MNR[c] (acres)	Ontario Hydro (miles)	
	Ground	Total	Aerial[d]	Ground
1948				6
1949				21
1950				
1951				325
1952		3		664
1953	7331	30	53	1134
1954		0	129	1248
1955		0	25	1678
1956	7475	2098	77	1670
1957		5579	425	3087
1958	5669	2161	482	3366
1959	6217	3389	408	3155
1960		1857	523	3571
1961	7169	4408	429	3794
1962	8193	2237	348	3495
1963	10,705	5207	278	3702
1964	11,421	5113	307	4893
1965	10,135	12,184	387	3483
1966	11,500	6978	358	3219
1967	14,665	7374	500	4500
1968	14,196	18,623	500	4500
1969	14,191	13,310	500	4500
1970	15259	385	500	4500
1971		2297	500	4500
1972	14,489	640	500	4500
1973	14,489	2499	500	4500
1974		499	500	4500
1975		25	500	4500
1976		133	500	4500
1977		4878	500	4500
1978		4220	500	4500
1979		19,477	500	4500

[a] *Blank cells = data not available and/or no spraying occurred.* [b] *MTO = Ministry of Transportation.*
[c] *MNR = Ministry of Natural Resources.* [d] *Assumed 10% where not reported.*

4.4. Chemicals of concern

Many herbicide products and formulations were used in Ontario; however, the concerns addressed in this assessment relate only to the use of 2,4,5-T, either alone or in combination with other herbicide products. The panel also considered TCDD, the known contaminant of 2,4,5-T.

4.4.1. Physical/chemical properties of 2,4,5-T and TCDD

Table 4.11 presents a summary of relevant physical/chemical properties for the chemicals of concern.

Table 4.11. *Physical/chemical properties for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Physical/chemical property[b]	2,4,5-T	TCDD
Molecular weight (g/mol)	255.49	322
Log K_{ow}	3.13	6.8
Melting point (°C)	153	305
Henry's Law Constant (Pa m^3/mol)	5.81e-3	3.337
Vapour pressure (Pa)	0.0922	1.18e-4
Water solubility (mg/L)	220	1.93e-5
Density (g/cm^3)	1.8	1.827
Soil half-life (days)	10-20	365-1095
Log-bioconcentration factor fish (L/kg)	NA	3.67 (rainbow trout; muscle)
BSAF-fish	NA	0.09
BTF beef (days/kg FW)	NA	1.58e-1
Soil-to-wet plant uptake factor	0.0926	8.76e-4
Partitioning factor (water)	NA	0.0138
Partitioning factor (sediment)	NA	0.97728
Soil-to-forage uptake factor	NA	0.0046
Reference	Mackay et al. 1997 US DE and ORO 2012	Mackay et al. 1992 US DE and ORO 2012 US EPA 2005

[a] *NA = Not applicable.*
[b] *In this column, BSAF = biota-to-sediment accumulation factor; BTF = bio-transfer factor; FW = fresh weight.*

4.4.2. Contaminant levels for 2,4,5-T

As discussed elsewhere, the 2,4,5-T used in Ontario contained manufacturing impurities (especially TCDD). Prior to regulation, the panel based contaminant level estimates on information available for 2,4,5-T stocks used in Vietnam. The panel assumed that once regulated, contaminant levels fell within those deemed acceptable through the pesticide registration process at the time of use (Table 4.12).

Table 4.12. *2,4,5-trichlorophenoxyacetic acid (2,4,5-T) contaminant levels, expressed as µg/g (parts per million; ppm) of active ingredient.*

Year range	Active ingredient	Contaminant	Contaminant level (µg/g; ppm)			Reference
			Low[a]	Central	High	
1948-1971	2,4,5-T	TCDD	0.05	13	47	Stellman et al. 2003; A0129372; A0138404
1972-1973	2,4,5-T	TCDD	0.5	0.5	0.5	A0129372; A0138404
1974-1979	2,4,5-T	TCDD	0.1	0.1	0.1	A0138404

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

4.4.3. Application rates for 2,4,5-T and TCDD

Several documents provided Ontario government users with direction on herbicide formulation concentrations and application rates. Section 4.3 describes these documents and the assumptions used to estimate herbicide application rates for this evaluation. The panel estimated contaminant (TCDD) application rates using the information in Table 4.12 and the following equation:

$$AR_{TCDD} = AI \times [TCDD] \qquad \text{(Equation 4.1)}$$

Where:

AR_{TCDD} = application rate of TCDD (units as below; Table 4.13)
AI = 2,4,5-T application (units as below; Table 4.13)
[TCDD] = TCDD level in 2,4,5-T (ppm; Table 4.12)

Table 4.13 summarizes the application rates used in this assessment.

Table 4.13. *Application rates for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) used for exposure assessment.*

Year range Ministry/agency	Product	Unit	Application rate			
			2,4,5-T	TCDD		
				Low[a]	Central	High
1948-1971						
Ministry of Natural Resources	2,4,5-T alone	lbs/acre	1.5	7.5E-08	2.0E-05	7.1E-05
	premix	lbs/acre	1	5.0E-08	1.3E-05	4.7E-05
Ontario Hydro	2,4,5-T alone	lbs/acre	3.5	1.8E-07	4.6E-05	1.6E-04
	premix	lbs/acre	1.75	8.8E-08	2.3E-05	8.2E-05
Ministry of Transportation	2,4,5-T alone	lbs/acre	1.5	7.5E-08	2.0E-05	7.1E-05
	premix[b]	lbs/acre	0.75	3.8E-08	9.8E-06	3.5E-05
1972-1973						
Ministry of Natural Resources	2,4,5-T alone	lbs/acre	1.5	7.5E-08	2.0E-05	7.1E-05
	premix	lbs/acre	1	5.0E-08	1.3E-05	4.7E-05
Ontario Hydro	2,4,5-T alone	lbs/acre	3.5	1.8E-06	1.8E-06	1.8E-06
	premix	lbs/acre	1.75	8.8E-07	8.8E-06	8.8E-06
Ministry of Transportation	2,4,5-T alone	lbs/acre	1.5	7.5E-07	7.5E-07	7.5E-07
	premix	lbs/acre	0.75	3.8E-07	3.8E-07	3.8E-07
1974-1979						
Ministry of Natural Resources	2,4,5-T alone	lbs/acre	1.5	7.5E-08	2.0E-05	7.1E-05
	premix	lbs/acre	1	5.0E-08	1.3E-05	4.7E-05
Ministry of Transportation	2,4,5-T alone	lbs/acre	3.5	3.5E-07	3.5E-07	3.5E-07
	premix	lbs/acre	1.75	1.8E-07	1.8E-07	1.8E-07

[a] Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.
[b] Premix represents situations when 2,4,5-T was used in a 50/50 combination with 2,4-D.

4.4.4. Environmental media concentrations for 2,4,5-T and TCDD

To predict environmental media concentrations (e.g., soil, plants, wild berries, etc.) of 2,4,5-T and TCDD resulting from herbicide use, the panel assumed minimum (min), central, and maximum (max) application rates for residential (ground spray) and remote (aerial spray) areas (see Section 4.4.3). Predicted soil concentrations were then used to estimate levels of each substance in wild berries, plants, and wildlife.

Two scenarios were considered for estimates of soil, plant, and wild berry concentrations:

- Residential:
  - Ontario Hydro: one day of ground spray, once every five years from 1948 to 1979, with property located 40 m from Ontario Hydro right of way (100 foot setback)
  - MTO: one day of ground spray, year after year from 1954 to 1973, with property located 20 m from MTO road (30 foot setback)
- Remote:
  - MNR: one season of aerial spray (max year) in remote area with intercept and drift loss
  - Ontario Hydro: one day of aerial spray, once every five years from 1948 to 1979, with intercept and drift loss

Table 4.14 provides the exposure parameter assumptions used to estimate media concentrations.

Table 4.14. *Exposure parameter assumptions used to estimate media concentrations of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Parameter	Unit	Value	Reference
Particulate concentration in air	kg/m^3	7.6×10^{-10}	Heath Canada 2004
Soil bulk density	kg/m^3	1500	US EPA 2005
Soil mixing zone—direct contact	m	0.01	US EPA 2005
Soil mixing zone—plant uptake	m	0.20	US EPA 2005
Sediment bulk density	kg/m^3	1500	Assumed
Sediment mixing zone	m	0.1	Assumed
Fraction reaching forest floor—central	unitless	0.12	Kreutzweiser and Nicholson 2007
Lipid content of fish	unitless	0.05	US EPA 2005
Organic carbon content of sediment	unitless	0.04	US EPA 2005

4.4.4.1. Aerial drift of herbicides

Residential (ground)

Residences were not located within the herbicide spray area but at some distance (setback) from the intended spray zone. For the Ontario Hydro scenario, residential properties were assumed to be located no closer than 40 m from the right of way. Similarly, for the MTO scenario, properties were assumed to be located no closer than 20 m from the roadway.

Remote (aerial)

Recreational areas were possibly affected by MNR spray activities. For Ontario Hydro, the panel assumed that recreational areas were set back at least 100 m from the right of way.

A standard drift model (AgDrift) and generic drift curves were used to estimate aerial off-target herbicide drift loss and related off-site herbicide deposition rates, which were assumed to contact the soil directly at the residential and recreational properties (Table 4.15).

4.4.4.2. Intercept fraction

For aerial spraying in remote areas, the panel assumed that the vegetation canopy would have intercepted some of the herbicide before it reached the soil. Kreutzweiser and Nicholson (2007) provided data to determine appropriate deposition fractions for forest canopies from approximately 205 field trials involving aerial spraying of insecticides over forested and open water areas. Based on their information, deposition fractions used in the current assessment have a central tendency estimate of 12%. However, insecticides are sprayed using finer droplets to maximize their penetration into the upper canopy; herbicides are generally applied using a medium to coarse droplet so they will penetrate to the lower canopy and/or forest floor. Thus, the deposition fractions derived from Kreutzweiser and Nicholson's (2007) data could underestimate actual percent deposit values for herbicides. This factor was applied only to aerial use, which was assumed to occur in non-residential/remote areas.

Table 4.15. *Predicted drift loss fractions used to estimate media concentrations of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Generic drift assumptions (worst case wind and spray nozzle)	Ground (boom)	Aerial-fixed	Aerial-rotary
Metres downwind	Percentage of target rate		
0	100%	100%	100%
1	3%	77%	
10			
20	4%		
30	3%		
40	2%		
50	1.5%		
100	1%	30%	
200		18%	6%
300		15%	3%
400		10%	2%
500		8%	1.5%
600		7%	1.25%
700		6%	1%
800		5%	1%

[a] *Blank cells = information not available.*

4.4.4.3. Calculating 2,4,5-T and TCDD concentrations in soil

TCDD and 2,4,5-T concentrations in soil were predicted for the MNR (remote), MTO (residential), and Ontario Hydro (remote and residential) spray scenarios using the methods in the U.S. Environmental Protection Agency Risk Assessment Protocol for Hazardous Waste Combustion Facilities (US EPA 2005) in combination with various estimates of chemical-specific soil half-life data and the first-order rate law. See Table 4.11 for the range of soil half-life values used for TCDD and 2,4,5-T.

As per U.S. EPA (2005), a soil mixing zone of 1 cm was assumed for soils that were available for direct contact (i.e., incidental soil ingestion and/or dermal contact) while a mixing zone of 20 cm was used to represent the root zone of wild plants. Concentrations of TCDD and 2,4,5-T in surface soil (at 1 cm and 20 cm in depth) were predicted. As previously indicated, drift loss fractions and a range of canopy intercept fractions were used to derive soil concentrations (recreational only). The following equation was used to predict soil concentrations after application for each scenario:

$$Soil_{initial} = \frac{DEP \times FCI \times DL \times CF}{BD \times MZ} \qquad \text{(Equation 4.2)}$$

Where:

$Soil_{initial}$	=	initial soil concentration immediately after spraying (µg/g; mg/kg)
DEP	=	application rate (lbs/acre)
CF	=	conversion factor (1 lb/acre = 112.1 mg/m^2)
FCI × DL	=	fraction of application rate making contact with soil surface (canopy intercept fraction × drift loss)
BD	=	soil bulk density (1500 kg/m^3)
MZ	=	soil mixing zone (0.01 m for direct contact; 0.2 m root zone)

For each area of concern, the panel calculated TCDD and 2,4,5-T soil concentrations immediately after an initial spray as shown above. For later applications (in the same area of concern), the initial soil concentration was degraded (using the first-order rate law and the half-lives) by the number of years between the first and second applications (assumed annually for MTO, every fifth year for Ontario Hydro; single application only for MNR). For MNR, only the maximum aerial application rate was considered, due to the single application and the focused nature of MNR ground applications (hand spray). This concentration was added to the soil concentration resulting from the second application and so forth. This process continued for all applications, and soil concentrations were predicted until 1989 (10 years after the last 2,4,5-T application). See tables 4.16 to 4.19 for the predicted TCDD and 2,4,5-T soil concentrations for each spray scenario.

$$Soil_{year\,n} = Soil_{initial} \times e^{-kt} \qquad \text{(Equation 4.3)}$$

Where:

$Soil_{year\,n}$	=	soil concentration *n* years after spraying (µg/g)
$Soil_{initial}$	=	initial soil concentration immediately after spraying
k	=	soil loss constant ($\ln 2/t_{1/2}$ where $t_{1/2}$ is the half-life of chemical in soil; d^{-1})
t	=	n years (days)

Table 4.16. *Predicted residential soil concentrations of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) following ground application (Ontario Hydro).*

Year	Surface soil (µg/g)				Subsurface soil (µg/g)			
	2,4,5-T	TCDD			2,4,5-T	TCDD		
		Low[a]	Central	High		Low	Central	High
1948	**5.23E-01**[b]	**2.62E-08**	**6.80E-06**	**2.46E-05**	**2.62E-02**	**1.31E-09**	**3.40E-07**	**1.23E-06**
1949	2.47E-08	1.31E-08	4.81E-06	1.95E-05	1.24E-09	6.54E-10	2.40E-07	9.76E-07
1950	1.17E-15	6.54E-09	3.40E-06	1.55E-05	5.85E-17	3.27E-10	1.70E-07	7.74E-07
1951	5.54E-23	3.27E-09	2.40E-06	1.23E-05	2.77E-24	1.63E-10	1.20E-07	6.15E-07
1952	2.62E-30	1.63E-09	1.70E-06	9.76E-06	1.31E-31	8.17E-11	8.50E-08	4.88E-07
1953	**5.23E-01**	**2.70E-08**	**8.00E-06**	**3.23E-05**	**2.62E-02**	**1.35E-09**	**4.00E-07**	**1.62E-06**
1954	2.47E-08	1.35E-08	5.66E-06	2.57E-05	1.24E-09	6.74E-10	2.83E-07	1.28E-06
1955	1.17E-15	6.74E-09	4.00E-06	2.04E-05	5.85E-17	3.37E-10	2.00E-07	1.02E-06
1956	5.54E-23	3.37E-09	2.83E-06	1.62E-05	2.77E-24	1.69E-10	1.41E-07	8.08E-07
1957	2.62E-30	1.69E-09	2.00E-06	1.28E-05	1.31E-31	8.43E-11	1.00E-07	6.41E-07
1958	**5.23E-01**	**2.70E-08**	**8.21E-06**	**3.48E-05**	**2.62E-02**	**1.35E-09**	**4.11E-07**	**1.74E-06**
1959	2.47E-08	1.35E-08	5.81E-06	2.76E-05	1.24E-09	6.75E-10	2.90E-07	1.38E-06
1960	1.17E-15	6.75E-09	4.11E-06	2.19E-05	5.85E-17	3.37E-10	2.05E-07	1.10E-06
1961	5.54E-23	3.37E-09	2.90E-06	1.74E-05	2.77E-24	1.69E-10	1.45E-07	8.69E-07
1962	2.62E-30	1.69E-09	2.05E-06	1.38E-05	1.31E-31	8.44E-11	1.03E-07	6.90E-07
1963	**5.23E-01**	**2.70E-08**	**8.25E-06**	**3.55E-05**	**2.62E-02**	**1.35E-09**	**4.13E-07**	**1.78E-06**
1964	2.47E-08	1.35E-08	5.83E-06	2.82E-05	1.24E-09	6.75E-10	2.92E-07	1.41E-06
1965	1.17E-15	6.75E-09	4.13E-06	2.24E-05	5.85E-17	3.37E-10	2.06E-07	1.12E-06
1966	5.54E-23	3.37E-09	2.92E-06	1.78E-05	2.77E-24	1.69E-10	1.46E-07	8.88E-07
1967	2.62E-30	1.69E-09	2.06E-06	1.41E-05	1.31E-31	8.44E-11	1.03E-07	7.05E-07
1968	**5.23E-01**	**2.70E-08**	**8.26E-06**	**3.58E-05**	**2.62E-02**	**1.35E-09**	**4.13E-07**	**1.79E-06**
1969	2.47E-08	1.35E-08	5.84E-06	2.84E-05	1.24E-09	6.75E-10	2.92E-07	1.42E-06
1970	1.17E-15	6.75E-09	4.13E-06	2.25E-05	5.85E-17	3.37E-10	2.06E-07	1.13E-06
1971	5.54E-23	3.37E-09	2.92E-06	1.79E-05	2.77E-24	1.69E-10	1.46E-07	8.94E-07
1972	2.62E-30	1.69E-09	2.06E-06	1.42E-05	1.31E-31	8.44E-11	1.03E-07	7.10E-07
1973	**5.23E-01**	**2.62E-07**	**1.72E-06**	**1.15E-05**	**2.62E-02**	**1.31E-08**	**8.61E-08**	**5.77E-07**
1974	2.47E-08	1.31E-07	1.22E-06	9.15E-06	1.24E-09	6.56E-09	6.09E-08	4.58E-07
1975	1.17E-15	6.56E-08	8.61E-07	7.26E-06	5.85E-17	3.28E-09	4.30E-08	3.63E-07
1976	5.54E-23	3.28E-08	6.09E-07	5.77E-06	2.77E-24	1.64E-09	3.04E-08	2.88E-07
1977	2.62E-30	1.64E-08	4.30E-07	4.58E-06	1.31E-31	8.20E-10	2.15E-08	2.29E-07
1978	**5.23E-01**	**6.05E-08**	**3.57E-07**	**3.68E-06**	**2.62E-02**	**3.03E-09**	**1.78E-08**	**1.84E-07**
1979	2.47E-08	3.03E-08	2.52E-07	2.92E-06	1.24E-09	1.51E-09	1.26E-08	1.46E-07
1980	1.17E-15	1.51E-08	1.78E-07	2.32E-06	5.85E-17	7.56E-10	8.92E-09	1.16E-07
1981	5.54E-23	7.56E-09	1.26E-07	1.84E-06	2.77E-24	3.78E-10	6.30E-09	9.21E-08
1982	2.62E-30	3.78E-09	8.92E-08	1.46E-06	1.31E-31	1.89E-10	4.46E-09	7.31E-08
1983	1.24E-37	1.89E-09	6.30E-08	1.16E-06	6.20E-39	9.45E-11	3.15E-09	5.80E-08
1984	5.86E-45	9.45E-10	4.46E-08	9.21E-07	2.93E-46	4.73E-11	2.23E-09	4.61E-08

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[b] ***Bold font*** *= application event.*

Table 4.17. *Predicted residential soil concentrations of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) following ground application (Ministry of Transportation).*[a]

Year	Surface soil (µg/g)				Subsurface soil (µg/g)			
	2,4,5-T	TCDD			2,4,5-T	TCDD		
		Low[b]	Central	High		Low	Central	High
1948								
1949								
1950								
1951								
1952								
1953								
1954	**4.51E-01**[c]	2.25E-08	5.86E-06	2.12E-05	**2.25E-02**	1.13E-09	2.93E-07	1.06E-06
1955	**4.51E-01**	**3.38E-08**	**1.00E-05**	**3.80E-05**	**2.25E-02**	1.69E-09	5.00E-07	1.90E-06
1956	**4.51E-01**	3.94E-08	1.29E-05	5.14E-05	**2.25E-02**	1.97E-09	6.47E-07	2.57E-06
1957	**4.51E-01**	**4.23E-08**	**1.50E-05**	**6.20E-05**	**2.25E-02**	2.11E-09	7.50E-07	3.10E-06
1958	**4.51E-01**	4.37E-08	1.65E-05	7.04E-05	**2.25E-02**	2.18E-09	8.24E-07	3.52E-06
1959	**4.51E-01**	**4.44E-08**	**1.75E-05**	**7.70E-05**	**2.25E-02**	2.22E-09	8.75E-07	3.85E-06
1960	**4.51E-01**	4.47E-08	1.82E-05	8.23E-05	**2.25E-02**	2.24E-09	9.12E-07	4.12E-06
1961	**4.51E-01**	**4.49E-08**	**1.88E-05**	**8.65E-05**	**2.25E-02**	2.25E-09	9.38E-07	4.33E-06
1962	**4.51E-01**	4.50E-08	1.91E-05	8.99E-05	**2.25E-02**	2.25E-09	9.56E-07	4.49E-06
1963	**4.51E-01**	**4.50E-08**	**1.94E-05**	**9.25E-05**	**2.25E-02**	2.25E-09	9.69E-07	4.63E-06
1964	**4.51E-01**	4.51E-08	1.96E-05	9.46E-05	**2.25E-02**	2.25E-09	9.78E-07	4.73E-06
1965	**4.51E-01**	**4.51E-08**	**1.97E-05**	**9.63E-05**	**2.25E-02**	2.25E-09	9.85E-07	4.81E-06
1966	**4.51E-01**	4.51E-08	1.98E-05	9.76E-05	**2.25E-02**	2.25E-09	9.89E-07	4.88E-06
1967	**4.51E-01**	**4.51E-08**	**1.99E-05**	**9.87E-05**	**2.25E-02**	2.25E-09	9.93E-07	4.93E-06
1968	**4.51E-01**	4.51E-08	1.99E-05	9.95E-05	**2.25E-02**	2.25E-09	9.95E-07	4.98E-06
1969	**4.51E-01**	**4.51E-08**	**1.99E-05**	**1.00E-04**	**2.25E-02**	2.25E-09	9.97E-07	5.01E-06
1970	**4.51E-01**	4.51E-08	2.00E-05	1.01E-04	**2.25E-02**	2.25E-09	9.98E-07	5.03E-06
1971	**4.51E-01**	**2.48E-07**	**1.43E-05**	**8.01E-05**	**2.25E-02**	1.24E-08	7.17E-07	4.01E-06
1972	**4.51E-01**	3.49E-07	1.04E-05	6.38E-05	**2.25E-02**	1.75E-08	5.18E-07	3.19E-06
1973	**4.51E-01**	**4.00E-07**	**7.55E-06**	**5.09E-05**	**2.25E-02**	2.00E-08	3.78E-07	2.54E-06
1974	2.13E-08	2.00E-07	5.34E-06	4.04E-05	1.07E-09	1.00E-08	2.67E-07	2.02E-06
1975	1.01E-15	1.00E-07	3.78E-06	3.21E-05	5.05E-17	5.00E-09	1.89E-07	1.60E-06
1976	4.77E-23	5.00E-08	2.67E-06	2.54E-05	2.39E-24	2.50E-09	1.34E-07	1.27E-06
1977	2.26E-30	2.50E-08	1.89E-06	2.02E-05	1.13E-31	1.25E-09	9.44E-08	1.01E-06
1978	1.07E-37	1.25E-08	1.34E-06	1.60E-05	5.34E-39	6.25E-10	6.68E-08	8.01E-07
1979	5.05E-45	6.25E-09	9.44E-07	1.27E-05	2.53E-46	3.13E-10	4.72E-08	6.36E-07
1980	2.39E-52	3.13E-09	6.68E-07	1.01E-05	1.20E-53	1.56E-10	3.34E-08	5.05E-07
1981	1.13E-59	1.56E-09	4.72E-07	8.01E-06	5.66E-61	7.81E-11	2.36E-08	4.01E-07
1982	5.35E-67	7.81E-10	3.34E-07	6.36E-06	2.68E-68	3.91E-11	1.67E-08	3.18E-07
1983	2.53E-74	3.91E-10	2.36E-07	5.05E-06	1.27E-75	1.95E-11	1.18E-08	2.52E-07
1984	1.20E-81	1.95E-10	1.67E-07	4.01E-06	5.99E-83	9.77E-12	8.34E-09	2.00E-07

[a] *Blank cells = data not available and/or no spraying occurred.*
[b] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[c] ***Bold font*** *= application event.*

Table 4.18. *Predicted remote soil concentrations of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) following aerial application (Ontario Hydro).*

Year	Surface soil (µg/g)				Subsurface soil (µg/g)			
	2,4,5-T	TCDD			2,4,5-T	TCDD		
		Low[a]	Central	High		Low	Central	High
1948	**9.41E-01**[b]	**4.71E-08**	**1.22E-05**	**4.42E-05**	**4.71E-02**	**2.35E-09**	**6.12E-07**	**2.21E-06**
1949	4.45E-08	2.35E-08	8.65E-06	3.51E-05	2.23E-09	1.18E-09	4.33E-07	1.76E-06
1950	2.11E-15	1.18E-08	6.12E-06	2.79E-05	1.05E-16	5.88E-10	3.06E-07	1.39E-06
1951	9.97E-23	5.88E-09	4.33E-06	2.21E-05	4.98E-24	2.94E-10	2.16E-07	1.11E-06
1952	4.72E-30	2.94E-09	3.06E-06	1.76E-05	2.36E-31	1.47E-10	1.53E-07	8.78E-07
1953	**9.41E-01**	**4.85E-08**	**1.44E-05**	**5.67E-05**	**4.71E-02**	**2.43E-09**	**7.20E-07**	**2.91E-06**
1954	4.45E-08	2.43E-08	1.02E-05	4.50E-05	2.23E-09	1.21E-09	5.09E-07	2.31E-06
1955	2.11E-15	1.21E-08	7.20E-06	3.57E-05	1.05E-16	6.07E-10	3.60E-07	1.83E-06
1956	9.97E-23	6.07E-09	5.09E-06	2.83E-05	4.98E-24	3.03E-10	2.55E-07	1.45E-06
1957	4.72E-30	3.03E-09	3.60E-06	2.25E-05	2.36E-31	1.52E-10	1.80E-07	1.15E-06
1958	**9.41E-01**	**4.86E-08**	**1.48E-05**	**6.21E-05**	4.71E-02	2.43E-09	7.39E-07	3.13E-06
1959	4.45E-08	2.43E-08	1.05E-05	4.93E-05	2.23E-09	1.21E-09	5.23E-07	2.48E-06
1960	2.11E-15	1.21E-08	7.39E-06	3.91E-05	1.05E-16	6.07E-10	3.70E-07	1.97E-06
1961	9.97E-23	6.07E-09	5.23E-06	3.10E-05	4.98E-24	3.04E-10	2.61E-07	1.56E-06
1962	4.72E-30	3.04E-09	3.70E-06	2.46E-05	2.36E-31	1.52E-10	1.85E-07	1.24E-06
1963	**9.41E-01**	**4.86E-08**	**1.49E-05**	**6.38E-05**	**4.71E-02**	**2.43E-09**	**7.43E-07**	**3.20E-06**
1964	4.45E-08	2.43E-08	1.05E-05	5.06E-05	2.23E-09	1.21E-09	5.25E-07	2.54E-06
1965	2.11E-15	1.21E-08	7.43E-06	4.02E-05	1.05E-16	6.07E-10	3.71E-07	2.01E-06
1966	9.97E-23	6.07E-09	5.25E-06	3.19E-05	4.98E-24	3.04E-10	2.63E-07	1.60E-06
1967	4.72E-30	3.04E-09	3.71E-06	2.53E-05	2.36E-31	1.52E-10	1.86E-07	1.27E-06
1968	**9.41E-01**	**4.86E-08**	**1.49E-05**	**6.43E-05**	4.71E-02	2.43E-09	7.43E-07	3.22E-06
1969	4.45E-08	2.43E-08	1.05E-05	5.11E-05	2.23E-09	1.21E-09	5.26E-07	2.56E-06
1970	2.11E-15	1.21E-08	7.43E-06	4.05E-05	1.05E-16	6.07E-10	3.72E-07	2.03E-06
1971	9.97E-23	6.07E-09	5.26E-06	3.22E-05	4.98E-24	3.04E-10	2.63E-07	1.61E-06
1972	4.72E-30	3.04E-09	3.72E-06	2.55E-05	2.36E-31	1.52E-10	1.86E-07	1.28E-06
1973	**9.41E-01**	**4.72E-07**	**3.10E-06**	**2.07E-05**	**4.71E-02**	**2.36E-08**	**1.55E-07**	**1.04E-06**
1974	4.45E-08	2.36E-07	2.19E-06	1.65E-05	2.23E-09	1.18E-08	1.10E-07	8.24E-07
1975	2.11E-15	1.18E-07	1.55E-06	1.31E-05	1.05E-16	5.90E-09	7.75E-08	6.54E-07
1976	9.97E-23	5.90E-08	1.10E-06	1.04E-05	4.98E-24	2.95E-09	5.48E-08	5.19E-07
1977	4.72E-30	2.95E-08	7.75E-07	8.23E-06	2.36E-31	1.48E-09	3.87E-08	4.12E-07
1978	**9.41E-01**	**1.09E-07**	**6.42E-07**	**6.63E-06**	4.71E-02	5.45E-09	3.21E-08	3.32E-07
1979	4.45E-08	5.45E-08	4.54E-07	5.26E-06	2.23E-09	2.72E-09	2.27E-08	2.63E-07
1980	2.11E-15	2.72E-08	3.21E-07	4.17E-06	1.05E-16	1.36E-09	1.60E-08	2.09E-07
1981	9.97E-23	1.36E-08	2.27E-07	3.31E-06	4.98E-24	6.81E-10	1.13E-08	1.66E-07
1982	4.72E-30	6.81E-09	1.60E-07	2.63E-06	2.36E-31	3.40E-10	8.02E-09	1.32E-07
1983	2.23E-37	3.40E-09	1.13E-07	2.09E-06	1.12E-38	1.70E-10	5.67E-09	1.04E-07
1984	1.06E-44	1.70E-09	8.02E-08	1.66E-06	5.28E-46	8.51E-11	4.01E-09	8.29E-08

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[b] ***Bold font*** *= application event.*

Table 4.19. *Predicted remote soil concentrations of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) following aerial application (Ministry of Natural Resources).*[a]

Year	Surface soil (µg/g)				Subsurface soil (µg/g)			
	2,4,5-T	TCDD			2,4,5-T	TCDD		
		Low[b]	Central	High		Low	Central	High
1948								
1949								
1950								
1951								
1952	**4.03E-01**[c]	**2.02E-08**	**5.25E-06**	**1.90E-05**	2.02E-02	1.01E-09	2.62E-07	9.48E-07
1953	4.15E-12	1.01E-08	3.71E-06	1.51E-05	2.08E-13	5.04E-10	1.85E-07	7.53E-07
1954	4.27E-23	5.04E-09	2.62E-06	1.19E-05	2.14E-24	2.52E-10	1.31E-07	5.97E-07
1955	4.40E-34	2.52E-09	1.85E-06	9.48E-06	2.20E-35	1.26E-10	9.27E-08	4.74E-07
1956	4.52E-45	1.26E-09	1.31E-06	7.53E-06	2.26E-46	6.30E-11	6.56E-08	3.76E-07
1957	4.65E-56	6.30E-10	9.27E-07	5.97E-06	2.33E-57	3.15E-11	4.64E-08	2.99E-07
1958	4.79E-67	3.15E-10	6.56E-07	4.74E-06	2.39E-68	1.58E-11	3.28E-08	2.37E-07
1959	4.93E-78	1.58E-10	4.64E-07	3.76E-06	2.46E-79	7.88E-12	2.32E-08	1.88E-07
1960	5.07E-89	7.88E-11	3.28E-07	2.99E-06	2.54E-90	3.94E-12	1.64E-08	1.49E-07
1961	5.22E-100	3.94E-11	2.32E-07	2.37E-06	2.61E-101	1.97E-12	1.16E-08	1.19E-07
1962	5.37E-111	1.97E-11	1.64E-07	1.88E-06	2.68E-112	9.85E-13	8.20E-09	9.41E-08
1963	5.52E-122	9.85E-12	1.16E-07	1.49E-06	2.76E-123	4.93E-13	5.80E-09	7.47E-08
1964	5.68E-133	4.93E-12	8.20E-08	1.19E-06	2.84E-134	2.46E-13	4.10E-09	5.93E-08
1965	5.85E-144	2.46E-12	5.80E-08	9.41E-07	2.92E-145	1.23E-13	2.90E-09	4.70E-08
1966	6.02E-155	1.23E-12	4.10E-08	7.47E-07	3.01E-156	6.16E-14	2.05E-09	3.73E-08
1967	6.19E-166	6.16E-13	2.90E-08	5.93E-07	3.10E-167	3.08E-14	1.45E-09	2.96E-08
1968	6.37E-177	3.08E-13	2.05E-08	4.70E-07	3.19E-178	1.54E-14	1.02E-09	2.35E-08
1969	6.56E-188	1.54E-13	1.45E-08	3.73E-07	3.28E-189	7.70E-15	7.24E-10	1.87E-08
1970	6.75E-199	7.70E-14	1.02E-08	2.96E-07	3.37E-200	3.85E-15	5.12E-10	1.48E-08
1971	6.94E-210	3.85E-14	7.24E-09	2.35E-07	3.47E-211	1.92E-15	3.62E-10	1.18E-08
1972	7.14E-221	1.92E-14	5.12E-09	1.87E-07	3.57E-222	9.62E-16	2.56E-10	9.33E-09
1973	7.35E-232	9.62E-15	3.62E-09	1.48E-07	3.68E-233	4.81E-16	1.81E-10	7.41E-09
1974	7.56E-243	4.81E-15	2.56E-09	1.18E-07	3.78E-244	2.41E-16	1.28E-10	5.88E-09
1975	7.78E-254	2.41E-15	1.81E-09	9.33E-08	3.89E-255	1.20E-16	9.06E-11	4.67E-09
1976	8.01E-265	1.20E-15	1.28E-09	7.41E-08	4.00E-266	6.01E-17	6.40E-11	3.70E-09
1977	8.24E-276	6.01E-16	9.06E-10	5.88E-08	4.12E-277	3.01E-17	4.53E-11	2.94E-09
1978	8.48E-287	3.01E-16	6.40E-10	4.67E-08	4.24E-288	1.50E-17	3.20E-11	2.33E-09
1979	8.73E-298	1.50E-16	4.53E-10	3.70E-08	4.36E-299	7.52E-18	2.26E-11	1.85E-09
1980	0.00E+00	7.52E-17	3.20E-10	2.94E-08	0.00E+00	3.76E-18	1.60E-11	1.47E-09
1981	0.00E+00	3.76E-17	2.26E-10	2.33E-08	0.00E+00	1.88E-18	1.13E-11	1.17E-09
1982	0.00E+00	1.88E-17	1.60E-10	1.85E-08	0.00E+00	9.39E-19	8.00E-12	9.26E-10
1983	0.00E+00	9.39E-18	1.13E-10	1.47E-08	0.00E+00	4.70E-19	5.66E-12	7.35E-10
1984	0.00E+00	4.70E-18	8.00E-11	1.17E-08	0.00E+00	2.35E-19	4.00E-12	5.83E-10

[a] *Blank cells = data not available and/or no spraying occurred.*
[b] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[c] ***Bold font*** *= application event.*

4.4.4.4. Calculating 2,4,5-T and TCDD concentrations in wild berries

Concentrations of TCDD and 2,4,5-T in wild berries were derived using the predicted soil concentration within the root zone (assuming a 20 cm mixing zone) and soil-to-plant uptake factors (Table 4.11). The following equation was used to predict wild berry concentrations:

$$C_{berries} = Soil \times UF_{plant} \qquad \text{(Equation 4.4)}$$

Where:

$C_{berries}$ = concentration of 2,4,5-T/TCDD in wild berries (µg/g)
Soil = concentration of 2,4,5-T/TCDD in soil at 20 cm depth (µg/g)
UF_{plant} = soil-to-plant uptake factor (unitless)

Concentrations of herbicides in wild berries predicted for areas sprayed by Ontario Hydro, MTO, and MNR are presented for residential areas in Table 4.20 and remote areas in Table 4.21.

Table 4.20. *Predicted concentration of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in wild berries (µg/g wet weight) in residential areas.*[a]

Year	Ontario Hydro				Ministry of Transportation			
	2,4,5-T	TCDD			2,4,5-T	TCDD		
		Low[b]	Central	High		Low	Central	High
1948	1.15E-12	2.98E-10	1.08E-09	1.15E-12				
1949	5.73E-13	2.11E-10	8.55E-10	5.73E-13				
1950	2.86E-13	1.49E-10	6.78E-10	2.86E-13				
1951	1.43E-13	1.05E-10	5.38E-10	1.43E-13				
1952	7.16E-14	7.45E-11	4.27E-10	7.16E-14				
1953	1.18E-12	3.50E-10	1.42E-09	1.18E-12				
1954	5.91E-13	2.48E-10	1.12E-09	5.91E-13	2.09E-03	9.87E-13	2.57E-10	9.28E-10
1955	2.95E-13	1.75E-10	8.92E-10	2.95E-13	2.09E-03	1.48E-12	4.38E-10	1.66E-09
1956	1.48E-13	1.24E-10	7.08E-10	1.48E-13	2.09E-03	1.73E-12	5.67E-10	2.25E-09
1957	7.38E-14	8.76E-11	5.62E-10	7.38E-14	2.09E-03	1.85E-12	6.57E-10	2.71E-09
1958	1.18E-12	3.60E-10	1.52E-09	1.18E-12	2.09E-03	1.91E-12	7.22E-10	3.08E-09
1959	5.91E-13	2.54E-10	1.21E-09	5.91E-13	2.09E-03	1.94E-12	7.67E-10	3.37E-09
1960	2.96E-13	1.80E-10	9.59E-10	2.96E-13	2.09E-03	1.96E-12	7.99E-10	3.61E-09
1961	1.48E-13	1.27E-10	7.61E-10	1.48E-13	2.09E-03	1.97E-12	8.22E-10	3.79E-09
1962	7.39E-14	8.99E-11	6.04E-10	7.39E-14	2.09E-03	1.97E-12	8.38E-10	3.94E-09
1963	1.18E-12	3.61E-10	1.56E-09	1.18E-12	2.09E-03	1.97E-12	8.49E-10	4.05E-09
1964	5.91E-13	2.56E-10	1.24E-09	5.91E-13	2.09E-03	1.97E-12	8.57E-10	4.14E-09
1965	2.96E-13	1.81E-10	9.80E-10	2.96E-13	2.09E-03	1.97E-12	8.63E-10	4.22E-09
1966	1.48E-13	1.28E-10	7.78E-10	1.48E-13	2.09E-03	1.97E-12	8.67E-10	4.28E-09
1967	7.39E-14	9.04E-11	6.18E-10	7.39E-14	2.09E-03	1.97E-12	8.70E-10	4.32E-09
1968	1.18E-12	3.62E-10	1.57E-09	1.18E-12	2.09E-03	1.97E-12	8.72E-10	4.36E-09
1969	5.91E-13	2.56E-10	1.24E-09	5.91E-13	2.09E-03	1.97E-12	8.73E-10	4.39E-09
1970	2.96E-13	1.81E-10	9.87E-10	2.96E-13	2.09E-03	1.97E-12	8.74E-10	4.41E-09
1971	1.48E-13	1.28E-10	7.83E-10	1.48E-13	2.09E-03	1.09E-11	6.28E-10	3.51E-09
1972	7.39E-14	9.04E-11	6.22E-10	7.39E-14	2.09E-03	1.53E-11	4.54E-10	2.80E-09
1973	1.15E-11	7.54E-11	5.05E-10	1.15E-11	2.09E-03	1.75E-11	3.31E-10	2.23E-09
1974	5.75E-12	5.33E-11	4.01E-10	5.75E-12	9.88E-11	8.76E-12	2.34E-10	1.77E-09
1975	2.87E-12	3.77E-11	3.18E-10	2.87E-12	4.67E-18	4.38E-12	1.65E-10	1.40E-09
1976	1.44E-12	2.67E-11	2.53E-10	1.44E-12	2.21E-25	2.19E-12	1.17E-10	1.11E-09
1977	7.18E-13	1.88E-11	2.00E-10	7.18E-13	1.05E-32	1.10E-12	8.27E-11	8.85E-10
1978	2.65E-12	1.56E-11	1.61E-10	2.65E-12	4.95E-40	5.48E-13	5.85E-11	7.02E-10
1979	1.33E-12	1.10E-11	1.28E-10	1.33E-12	2.34E-47	2.74E-13	4.14E-11	5.57E-10
1980	6.63E-13	7.81E-12	1.02E-10	6.63E-13	1.11E-54	1.37E-13	2.92E-11	4.42E-10
1981	3.31E-13	5.52E-12	8.07E-11	3.31E-13	5.24E-62	6.85E-14	2.07E-11	3.51E-10
1982	1.66E-13	3.90E-12	6.40E-11	1.66E-13	2.48E-69	3.42E-14	1.46E-11	2.79E-10
1983	8.28E-14	2.76E-12	5.08E-11	8.28E-14	1.17E-76	1.71E-14	1.03E-11	2.21E-10
1984	4.14E-14	1.95E-12	4.03E-11	4.14E-14	5.55E-84	8.56E-15	7.31E-12	1.76E-10

[a] *Blank cells = data not available and/or no spraying occurred.*
[b] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

Table 4.21. *Predicted concentrations of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in wild berries (µg/g wet weight) in remote areas.*[a]

Year	Ontario Hydro				Ministry of Natural Resources			
	2,4,5-T	TCDD			2,4,5-T	TCDD		
		Low[b]	Central	High		Low	Central	High
1948	4.36E-03	2.06E-12	5.36E-10	1.94E-09				
1949	2.06E-10	1.03E-12	3.79E-10	1.54E-09				
1950	9.76E-18	5.15E-13	2.68E-10	1.22E-09				
1951	4.62E-25	2.58E-13	1.90E-10	9.69E-10				
1952	2.18E-32	1.29E-13	1.34E-10	7.69E-10	1.87E-03	8.84E-13	2.30E-10	8.31E-10
1953	4.36E-03	2.13E-12	6.31E-10	2.55E-09	1.92E-14	4.42E-13	1.62E-10	6.59E-10
1954	2.06E-10	1.06E-12	4.46E-10	2.02E-09	1.98E-25	2.21E-13	1.15E-10	5.23E-10
1955	9.76E-18	5.32E-13	3.15E-10	1.61E-09	2.04E-36	1.10E-13	8.12E-11	4.15E-10
1956	4.62E-25	2.66E-13	2.23E-10	1.27E-09	2.09E-47	5.52E-14	5.74E-11	3.30E-10
1957	2.18E-32	1.33E-13	1.58E-10	1.01E-09	2.15E-58	2.76E-14	4.06E-11	2.62E-10
1958	4.36E-03	2.13E-12	6.48E-10	2.74E-09	2.22E-69	1.38E-14	2.87E-11	2.08E-10
1959	2.06E-10	1.06E-12	4.58E-10	2.18E-09	2.28E-80	6.90E-15	2.03E-11	1.65E-10
1960	9.76E-18	5.32E-13	3.24E-10	1.73E-09	2.35E-91	3.45E-15	1.44E-11	1.31E-10
1961	4.62E-25	2.66E-13	2.29E-10	1.37E-09	2.42E-102	1.73E-15	1.02E-11	1.04E-10
1962	2.18E-32	1.33E-13	1.62E-10	1.09E-09	2.49E-113	8.63E-16	7.18E-12	8.24E-11
1963	4.36E-03	2.13E-12	6.51E-10	2.80E-09	2.56E-124	4.31E-16	5.08E-12	6.54E-11
1964	2.06E-10	1.06E-12	4.60E-10	2.22E-09	2.63E-135	2.16E-16	3.59E-12	5.19E-11
1965	9.76E-18	5.32E-13	3.25E-10	1.76E-09	2.71E-146	1.08E-16	2.54E-12	4.12E-11
1966	4.62E-25	2.66E-13	2.30E-10	1.40E-09	2.79E-157	5.39E-17	1.79E-12	3.27E-11
1967	2.18E-32	1.33E-13	1.63E-10	1.11E-09	2.87E-168	2.70E-17	1.27E-12	2.60E-11
1968	4.36E-03	2.13E-12	6.51E-10	2.82E-09	2.95E-179	1.35E-17	8.97E-13	2.06E-11
1969	2.06E-10	1.06E-12	4.60E-10	2.24E-09	3.04E-190	6.74E-18	6.35E-13	1.64E-11
1970	9.76E-18	5.32E-13	3.26E-10	1.78E-09	3.12E-201	3.37E-18	4.49E-13	1.30E-11
1971	4.62E-25	2.66E-13	2.30E-10	1.41E-09	3.21E-212	1.69E-18	3.17E-13	1.03E-11
1972	2.18E-32	1.33E-13	1.63E-10	1.12E-09	3.31E-223	8.43E-19	2.24E-13	8.18E-12
1973	4.36E-03	2.07E-11	1.36E-10	9.09E-10	3.40E-234	4.21E-19	1.59E-13	6.49E-12
1974	2.06E-10	1.03E-11	9.60E-11	7.21E-10	3.50E-245	2.11E-19	1.12E-13	5.15E-12
1975	9.76E-18	5.17E-12	6.79E-11	5.73E-10	3.60E-256	1.05E-19	7.93E-14	4.09E-12
1976	4.62E-25	2.59E-12	4.80E-11	4.55E-10	3.71E-267	5.27E-20	5.61E-14	3.24E-12
1977	2.18E-32	1.29E-12	3.39E-11	3.61E-10	3.82E-278	2.63E-20	3.97E-14	2.58E-12
1978	4.36E-03	4.77E-12	2.81E-11	2.90E-10	3.93E-289	1.32E-20	2.80E-14	2.04E-12
1979	2.06E-10	2.39E-12	1.99E-11	2.31E-10	4.04E-300	6.58E-21	1.98E-14	1.62E-12
1980	9.76E-18	1.19E-12	1.41E-11	1.83E-10	0.00E+00	3.29E-21	1.40E-14	1.29E-12
1981	4.62E-25	5.96E-13	9.94E-12	1.45E-10	0.00E+00	1.65E-21	9.92E-15	1.02E-12
1982	2.18E-32	2.98E-13	7.03E-12	1.15E-10	0.00E+00	8.23E-22	7.01E-15	8.11E-13
1983	1.03E-39	1.49E-13	4.97E-12	9.15E-11	0.00E+00	4.11E-22	4.96E-15	6.44E-13
1984	4.89E-47	7.45E-14	3.51E-12	7.26E-11	0.00E+00	2.06E-22	3.51E-15	5.11E-13

[a] *Blank cells = data not available and/or no spraying occurred.*
[b] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

4.4.4.5. Calculating 2,4,5-T and TCDD concentrations in surface water, sediment, and fish

No data exist for historical contaminant concentrations in fish, sediment, or surface water for relevant years. While small watercourses and edges of ponds, reservoirs, and lakes were not deliberately sprayed, some overspray likely occurred. Similar to contaminant concentrations in surface soils and wild berries, water and sediment concentrations were predicted from historical application rates and some worst case assumptions made based on drift, canopy intercept, environmental partitioning, and water depth (as discussed in Solomon et al. 2005). TCDD's bioaccumulative nature was considered in this evaluation. Predicting historical water and sediment concentrations from historical contaminant application rates requires many assumptions and introduces many

uncertainties. Fish tissue concentrations were predicted based on estimated sediment concentrations in a remote area of Ontario that may have been inadvertently sprayed aerially (Ontario Hydro and MNR). Only direct spray was considered since TCDD is extremely hydrophobic—it tends to readily bind to soil and would not be expected to run off from the watershed area in significant quantities. A single MNR and/or Ontario Hydro spray event was considered. Sediment and water concentration were estimated as follows:

$$C_{water} = \frac{DEP \times FCI \times DL \times PF \times CF}{MZ} \qquad \text{(Equation 4.5)}$$

Where:

C_{water}	=	initial surface water concentration (µg/L)
DEP	=	application rate (lbs/acre)
CF	=	conversion factor (1 lb/acre = 112.1 mg/m^2)
FCI × DL	=	fraction of application rate making contact with soil surface (canopy intercept fraction × drift loss)
PF	=	partitioning factor (0.0138; portion of deposited TCDD that will remain in water)
MZ	=	water mixing zone (L/m^2)

$$C_{sediment} = \frac{DEP \times FCI \times DL \times PF \times CF}{BD \times MZ} \qquad \text{(Equation 4.6)}$$

Where:

$C_{sediment}$	=	initial sediment concentration (µg/g; mg/kg)
DEP	=	application rate (lbs/acre)
CF	=	conversion factor (1 lb/acre = 112.1 mg/m2)
FCI × DL	=	fraction of application rate making contact with soil surface (canopy intercept fraction × drift loss)
PF	=	partitioning factor (0.97728; portion of deposited TCDD that will partition to sediment)
BD	=	sediment bulk density (1,500 kg/m^3)
MZ	=	sediment mixing zone (0.1 m)

Table 4.22 presents the estimated water and sediment concentrations.

Table 4.22. *Estimated concentrations of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in surface water and sediment (µg/L).*

	Ontario Hydro			Ministry of Natural Resources		
	Low[a]	Central	High	Low	Central	High
Surface water (2 m deep, rapid mixing, no flow, and absorption to sediment)	4.87E-09	1.27E-06	4.58E-06	2.09E-09	5.43E-07	1.96E-06
Surface water (1 m deep, rapid mixing, no flow, and absorption to sediment)	9.74E-09	2.53E-06	9.16E-06	4.18E-09	1.09E-06	3.93E-06
Surface water (0.3 m deep, rapid mixing, no flow, and absorption to sediment)	3.25E-08	8.45E-06	3.05E-05	1.39E-08	3.62E-06	1.31E-05
Surface water (0.15 m deep, rapid mixing, no flow, and absorption to sediment)	6.50E-08	1.69E-05	6.11E-05	2.78E-08	7.24E-06	2.62E-05
Sediment (97.728% partitioning to sediment with water input)	4.60E-09	1.20E-06	4.32E-06	1.97E-09	5.13E-07	1.85E-06

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

It is preferable to use a biota-to-sediment accumulation factor (BSAF) when predicting TCDD fish tissue concentrations (US EPA 2005). The panel predicted fish concentrations (Table 4.23) using the following equation:

$$C_{fish} = \left(\frac{C_{sediment} \times f_{lipid} \times BSAF_{fish}}{OC_{sediment}} \right) \qquad \text{(Equation 4.7)}$$

Where:

C_{fish} = concentration of TCDD in fish tissue (µg/g fresh weight tissue)
$C_{sediment}$ = concentration of TCDD in sediment (µg/g)
f_{lipid} = lipid content of fish (0.07 unitless)
$BSAF_{fish}$ = biota-to-sediment accumulation factor (0.09 unitless)
$OC_{sediment}$ = organic carbon content of sediment (0.03 unitless)

Table 4.23. *Estimated concentrations of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in fish (µg/g).*

Ontario Hydro			Ministry of Natural Resources		
Low[a]	Central	High	Low	Central	High
9.66E-10[b]	2.51E-07	9.08E-07	4.14E-10	1.08E-07	3.89E-07

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[b] *Fish concentrations based on 97.728% partitioning to sediment with water input from Table 4.22.*

4.4.4.6. Calculating 2,4,5-T and TCDD concentrations in wild game

Hunters were assumed to spend time hunting in remote areas affected by aerial spraying (MNR and Ontario Hydro) and to consume wild game, i.e., moose (*Alces alces*) and white-tailed deer (*Odocoileus virginianus*) from these areas. Due to its bioaccumulative nature, only TCDD was considered in this evaluation. Before calculating TCDD exposures of individuals consuming wild game, the panel estimated TCDD concentrations in moose and white-tailed deer using the methods provided by Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (US EPA 2005). 2,4,5-T was not considered for this pathway since it does not bioaccumulate.

To determine concentrations of TCDD in tissues of wild game (i.e., moose and deer), an exposure assessment was first conducted to estimate the average daily dose of contaminants that moose and deer might get from the environment (i.e., water, soil, and vegetation). Moose and deer were assumed to spend all of their time in the vicinity of an aerial spraying (MNR and Ontario Hydro). Moose are generally considered a non-migratory species, although they will move seasonally to find optimum environmental conditions (Hundertmark 1997). Home range size is influenced by factors such as habitat quality, population parameters, and food availability (Hundertmark 1997). The home range of moose is about 26 km^2 for males and about 14 km^2 for females (Cederlund and Sand 1994). Female moose with young <12 months old have been observed to have a home range of approximately 2.2 km^2 (Cederlund and Sand 1994). Allen et al. (1987) reported that a moose home range is approximately 6 km^2, that the density of moose populations (assuming optimal habitat) is 0.4 to 4 moose/km^2, and that an area of 93 km^2 is sufficient to support a viable moose population over long periods.

The home range of white-tailed deer is often <1 km^2 (Dewey 2003). Their diet varies seasonally with food availability. In the spring and early summer, deer consume primarily shoots, leaves and twigs, shrubs, grasses, and forbs (Short 1986). During the summer and into the winter, fruits, seeds, and nuts become important sources of

food. Deciduous leaves are consumed until they die off in the fall, and coniferous leaves may be consumed all year (Short 1986). Cool season vegetation provides a highly digestible food source during the winter and early spring (Short 1986).

The objective of the wild game exposure assessment was to estimate the average daily dose of TCDD for moose and deer during the time 2,4,5-T was sprayed in Ontario. These data were used to predict TCDD concentrations in tissues of wild game. Because it is impossible to determine when an affected animal (receptor) might have been caught and eaten, it was important to capture the maximum daily dose over the entire time frame that herbicide spraying was conducted in these areas. As a result, a single maximum estimate of TCDD exposure in moose and deer was generated for each relevant spray scenario. Predicted water and soil concentrations (tables 4.18, 4.19, 4.22) were used to predict exposures via incidental soil ingestion and forage consumption. The panel assumed wild game were exposed to contaminants via incidental soil ingestion, forage consumption, and surface water ingestion.

Variables considered as part of the exposure assessment included:

- physical-chemical properties of TCDD that define its movement in the environment and transfer from one environmental compartment to another
- concentrations of TCDD in surface water
- relevant exposure pathways and exposure routes by which TCDD might travel from the affected soil and surface water to the wild game
- physical and behavioural characteristics of moose and deer (e.g., weight, body surface area, daily feed consumption, daily water consumption, home range, diet, etc.)

Table 4.24 lists the assumptions used to calculate herbicide concentrations in wild game in Ontario.

Table 4.24. *Assumptions used to calculate concentrations of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in wild game.*

Parameter	Wild game type	Unit	Value	Reference
Fraction of time spent in spray area	Moose	unitless	0.1	Assumed
	Deer	unitless	1	Assumed
Body weight	Moose	kg	353	Belovsky 1978
	Deer	kg	56.5	Sample and Suter 1994
Forage consumed	Moose	kg DW/day	7.5 (25)[a]	Tefler 1997
	Deer	kg DW/day	0.42 (1.74)	Sample and Suter 1994
Sediment/soil consumed	Moose	kg/day	0.15	Beyer et al. 1994
	Deer	kg/day	0.01	Arthur and Alldredge 1979

[a] *Values in parentheses are the amount of forage consumed (kg DW/day) converted to a dry weight (DW) assuming a 70% moisture content.*

Calculating wild game exposures

The exposure methodology used was recommended by the U.S. EPA Region 6 Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (US EPA 2005). Following is a summary of the methods used to approximate exposures of wild game to TCDD in remote areas of Ontario.

Exposure via consumption of forage

Aboveground vegetation growing on affected soil could accumulate contaminants from root uptake. Plant-to-soil bioconcentration factors for forage were provided by the U.S. EPA (2005) and were used to calculate forage concentrations based on predicted soil concentrations. The panel estimated TCDD concentration in forage as follows:

$$C_f = C_s \times B_f \qquad \text{(Equation 4.8)}$$

Where:

Cf	=	concentration in forage as a result of root uptake from soil (mg/kg dry weight)
C_s	=	predicted subsurface soil concentration (µg/g)
B_f	=	Soil-to-forage bioconcentration factor (unitless)

The panel estimated wild game exposure to TCDD by consuming contaminant-affected forage as follows:

$$EXP_f = \frac{C_f \times FT \times FCR}{BW} \qquad \text{(Equation 4.9)}$$

Where:

EXP_f	=	estimated daily intake via consuming forage (mg/kg BW/day)
C_f	=	predicted concentration of TCDD in forage (mg/kg dry weight)
FT	=	fraction of time in spray area (0.1 for moose due to its large home range; 1.0 for deer)
FCR	=	receptor-specific forage consumption rate (kg dry weight/day)
BW	=	receptor-specific body weight (kg)

Exposure via incidental ingestion of soil

White-tailed deer are likely to consume soil incidentally while foraging on forbs, seeds, nuts, grasses, and other foods. A study of mule deer (*Odocoileus hemionus*) showed winter, spring, summer, and fall soil intakes rates of 0.0183, 0.0296, 0.0077, and 0.0088 kg/day, respectively (Arthur and Alldredge 1979). This study also showed that soil intake rates ranged from 0.6 to 2% of the total food intake rate. The values in this study were extrapolated to derive a soil intake rate of 1.4% for white-tailed deer, which corresponds with a study on white-tailed deer that found that soil comprises <2% of total diet (Beyer et al. 1994). Moose likely consume minor amounts of soil intentionally for the salt content or incidentally while consuming vegetation. The panel assumed that moose consume <2% soil in their diet, as calculated by Beyer et al. (1994).

The panel estimated wild game exposure to TCDD via incidental ingestion of soil as follows:

$$EXP_{soil} = \frac{C_{soil} \times SIR \times FT \times BA_{soil}}{BW} \qquad \text{(Equation 4.10)}$$

Where:

EXP_{soil}	=	estimated daily intake from soil ingestion (mg/kg BW/day)
C_{soil}	=	predicted concentration in surface soil (µg/g)
BA_{soil}	=	bioavailability of TCDD in soil (unitless)
FT	=	fraction of time in spray area (0.1 for moose due to its large home range; 1.0 for deer)
SIR	=	receptor-specific soil ingestion rate (kg/day)
BW	=	receptor-specific body weight (kg)

Exposure to affected surface water

The panel also assumed wild game were exposed to TCDD via ingestion of affected surface water and estimated that as follows:

$$EXP_{sw} = \frac{C_{sw} \times BA_{sw} \times FT \times WIR}{BW} \qquad \text{(Equation 4.11)}$$

Where:

EXP_{sw}	=	estimated daily intake via ingestion of water (mg/kg BW/day)
C_{sw}	=	concentration of TCDD in surface water (mg/L)
BA_{sw}	=	bioavailability of TCDD in water (assumed 100%)
FT	=	fraction of time in spray area (0.1 for moose due to its large home range; 1.0 for deer)
WIR	=	receptor-specific water ingestion rate (L/day)
BW	=	receptor-specific body weight (kg)

Predicting wild game tissue concentrations

To predict tissue concentrations of TCDD in moose and white-tailed deer, the panel used chemical-specific bio-transfer factors (BTFs) that the Research Triangle Institute developed (RTI 2005) and the U.S. EPA used (US EPA 2005) to predict TCDD concentrations in beef cattle. These BTF values replace the older and most common methodology developed by Travis and Arms (1988), as their regression equations were considered limited due to the small range of log K_{ow} values used to develop each regression. The new methodology (RTI 2005) used an empirical data set with improved quantity and quality over the previous study (Travis and Arms 1988).

The tissue concentration of TCDD in wild game via consuming forage, soil, and surface water was estimated as follows:

$$Beef_{tissue} = EXP_{total} \times BTF_{beef} \times BW \qquad \text{(Equation 4.12)}$$

Where:

$Beef_{tissue}$	=	predicted concentration of TCDD in game tissue (mg/kg fresh weight)
EXP_{total}	=	predicted exposure of game to TCDD (mg/kg BW /day)
BW	=	receptor-specific body weight (kg)
BTF_{beef}	=	bio-transfer factor for beef cattle (day/kg fresh weight)

Table 4.25 summarizes the calculations and exposure parameters used to estimate TCDD concentrations in wild game tissues.

Table 4.25. *Parameters used to estimate concentrations of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in wild game tissues.*

	Ontario Hydro						
	Unit	Moose			White-tailed deer		
		Low[a]	Central	High	Low	Central	High
Receptor parameter							
Body weight	kg	353	353	353	56.5	56.5	56.5
Forage consumed	kg DW/day[b]	7.5	7.5	7.5	0.42	0.42	0.42
Fraction of forage/soil/water from spray area	unitless	0.1	0.1	0.1	1	1	1
Sediment/soil consumed	kg/day	0.15	0.15	0.15	0.01	0.01	0.01
Water consumed	L/day	19.44	19.44	19.44	3.74	3.74	3.74
Fraction of intake from area	unitless	1	1	1	1	1	1
Chemical parameter							
Soil concentration—ingestion	mg/kg	4.7E-07	1.5E-05	6.4E-05	4.7E-07	1.5E-05	6.4E-05
Soil concentration—plant uptake	mg/kg	2.4E-08	7.4E-07	3.2E-06	2.4E-08	7.4E-07	3.2E-06
Surface water concentration	µg/L	3.2E-08	8.4E-06	3.1E-05	3.2E-08	8.4E-06	3.1E-05
Biotransfer factor for forage	unitless	4.6E-03	4.6E-03	4.6E-03	4.6E-03	4.6E-03	4.6E-03
Beef biotransfer factor	day/kg FW[c]	1.6E-01	1.6E-01	1.6E-01	1.6E-01	1.6E-01	1.6E-01
Forage concentration	mg/kg DW	1.09E-10	3.4E-09	1.48E-08	1.1E-10	3.4E-09	1.5E-08
Soil bioavailability	unitless	3.4E-01	3.4E-01	3.4E-01	3.4E-01	3.4E-01	3.4E-01
Exposure estimate							
Forage consumption	mg/kg/day	2.3E-13	7.26E-12	3.15E-11	8.1E-13	2.5E-11	1.1E-10
Direct water ingestion	mg/kg/day	1.79E-13	4.65E-11	1.68E-10	2.15E-12	5.59E-10	2.02E-09
Incidental soil ingestion	mg/kg/day	6.82E-12	2.15E-10	9.30E-10	1.67E-11	5.26E-10	2.28E-09
Total exposure	mg/kg/day	7.2E-12	2.7E-10	1.13E-09	2.0E-11	1.1E-09	4.4E-09
Beef [tissue]	mg/kg FW[c]	4.0E-10	1.5E-08	6.3E-08	1.8E-10	9.9E-09	3.9E-08
	Ministry of Natural Resources						
	Unit	Moose			White-tailed deer		
		Low	Central	High	Low	Central	High
Receptor parameter							
Body weight	kg	353	353	353	56.5	56.5	56.5
Forage consumption	kg DW/day	7.5	7.5	7.5	0.42	0.42	0.42
Fraction of forage/soil/water from spray area	untiless	0.1	0.1	0.1	1	1	1
Sediment/soil consumed	kg/day	0.15	0.15	0.15	0.01	0.01	0.01
Water consumed	L/day	19.44	19.44	19.44	3.74	3.74	3.74
Fraction of intake from area	unitless	1	1	1	1	1	1
Chemical parameter							
Soil concentration—ingestion	mg/kg	2.0E-08	5.2E-06	1.9E-05	2.0E-08	5.2E-06	1.9E-05
Soil concentration—plant uptake	mg/kg	1.0E-09	2.6E-07	9.5E-07	1.0E-09	2.6E-07	9.5E-07
Surface water concentration	µg/L	1.4E-08	3.6E-06	1.3E-05	1.4E-08	3.6E-06	1.3E-05
Biotransfer factor for forage	unitless	4.6E-03	4.6E-03	4.6E-03	4.6E-03	4.6E-03	4.6E-03
Beef biotransfer factor	day/kg FW	1.6E-01	1.6E-01	1.6E-01	1.6E-01	1.6E-01	1.6E-01
Forage concentration	mg/kg DW	4.64E-12	1.2E-09	4.36E-09	4.6E-12	1.2E-09	4.4E-09
Soil bioavailability	unitless	3.4E-01	3.4E-01	3.4E-01	3.4E-01	3.4E-01	3.4E-01
Exposure estimate							
Forage consumption	mg/kg/day	9.9E-15	2.56E-12	9.27E-12	3.4E-14	9.0E-12	3.2E-11
Direct water ingestion	mg/kg/day	7.66E-14	1.99E-11	7.21E-11	9.21E-13	2.39E-10	8.65E-10
Incidental soil ingestion	mg/kg/day	2.91E-13	7.58E-11	2.74E-10	7.14E-13	1.86E-10	6.71E-10
Total exposure	mg/kg/day	3.8E-13	9.8E-11	3.55E-10	1.7E-12	4.3E-10	1.6E-09
Beef [tissue]	mg/kg FW	2.1E-11	5.5E-09	2.0E-08	1.5E-11	3.9E-09	1.4E-08

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[b] *DW = dry weight.*

[c] *FW = fresh weight.*

4.5. Individuals of potential concern

4.5.1. Selecting receptors

Since the quantitative risk assessment does not involve evaluating individuals case by case, the panel had to identify a series of receptor groups that can be used to represent all types of individuals. The following receptor groups were considered:

- Occupational
  - mixer/loader/applicator (ground)
    - backpack
    - tractor/truck
  - mixer/loader/applicator (pilot) (aerial)
    - flagman
    - supervisor
    - junior ranger
- Non-occupational
  - bystander
  - resident
  - recreational visitor/hunter/angler
  - family of worker
  - First Nations person (dietary)

Table 4.26 indicates which receptor groups were evaluated for each of the exposure scenarios discussed in Section 4.3.

Table 4.26. *Summary of receptor groups evaluated for exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Receptor group	Ministry of Natural Resources	Ontario Hydro	Ministry of Transportation
Worker			
• mixer/loader/applicator (ground)	X[b]	X	X
• mixer/loader/applicator/flagman (aerial)	X	X	
• scout/junior ranger	X		
Bystander	(x)[c]	(x)	(x)
Resident	(x)	(x)	(x)
Recreational visitor/hunter/angler	(x)		
Family member of worker	(x)	(x)	(x)
First Nations person (dietary)	(x)		

[a] *Blank cells = receptor not considered.*
[b] *X = significant pathway.*
[c] *(x) = minor pathway.*

Following is the list of receptor groups, including a description of how they might have encountered the chemicals of concern:

- Mixer/loaders:
  - Were responsible for handling, mixing, loading, and cleaning up herbicides. Did tasks such as preparing for an application using concentrated end-use products; for example, would prepare dilute spray solutions and/or load/transfer materials or dilute spray solutions into application equipment.
  - Could have been exposed to herbicides while handling, mixing, loading, and/or cleaning up during and after spraying. These tasks could have been divided among several individuals or teams, so no single person would have handled all the herbicide during spraying, but the lack of historical data required the panel to use a more conservative assumption.

- Were assumed to be exposed via both dermal absorption and inhalation.
- Were evaluated for both aerial and ground application methods.

- Applicators:
  - Were pilots, boom sprayer operators, and other field crew members who operated the application equipment during herbicide spraying.
  - Were assumed to be exposed via both dermal absorption and inhalation.
  - Were evaluated for both aerial and ground spraying application methods. Ground spraying was generally a more conservative exposure activity than was aerially applying herbicide.

- Flagmen:
  - Were responsible for marking the beginning or end of an aerial spray plot to guide aerial applicators during herbicide release onto an intended target.
  - Were primarily exposed to dilute spray.
  - Were assumed to be exposed via both dermal absorption and inhalation.
  - Could have been exposed to herbicide spray during each application.

- Junior rangers:
  - Accompanied MNR spray teams and participated in spray activities such as mixing, loading, and application.
  - Had unique age-related characteristics, which the panel considered where appropriate when calculating exposure.

- Supervisors:
  - Accompanied spray teams and oversaw spray activities such as mixing, loading, and application.
  - Were assumed to have been exposed similarly and/or less than others directly involved in herbicide spraying (i.e., mixers/loaders/applicators).

- Bystanders of aerial spray drift or overspray:
  - Were within or adjacent to an area where herbicide was being or had been applied. Their presence was incidental and unrelated to work involving herbicides, yet they may have been at risk of potential exposure, and they took no action to avoid or control exposure.
  - Were assumed to have worn no protective clothing
  - Were evaluated for exposures via both dermal absorption and inhalation at different distances from the spray block edge.

- Residents:
  - Lived, worked, or attended a school or other institution adjacent to an area treated with herbicide. Their presence was incidental and unrelated to work involving herbicide, yet they may have been at risk of potential exposure, and they took no action to avoid or control exposure.
  - Were assumed to have worn no protective clothing and might have been in the location for 24 hours a day. May have had direct contact with various environmental media (soil, dust, local produce such as

berries, etc.) affected by herbicide contaminants as part of their daily lives. Were assumed to live in a single location for 25 years.

- Recreational visitors:
  - Used remote areas for recreation so may have come into contact with soil or water in sprayed areas.
  - Were assumed to be adults who may have spent significant amounts of time in these areas during the summer months. Young children (6 months to 4 years old) were assumed to have been exposed in residential areas, as they were unlikely to spend significant amounts of time unsupervised at remote locations.

- Hunters/anglers:
  - May have used remote areas for hunting and fishing so may have been exposed to contaminants via consuming wild game and/or fish from sprayed areas.
  - Were assumed to be adults, but the panel also assumed that children of hunters/anglers consumed wild game and fish.

- First Nations people (dietary):
  - Similarly to the hunters/anglers, were assumed to use remote areas for hunting and fishing.
  - Were assumed to be subsistence hunters and anglers.

- Spouses/other family members:
  - May have been exposed to the chemicals of concern through contact with the clothing of family members involved in spray activities.
  - Were not quantitatively evaluated, but the panel did discuss this potential exposure route further.

Given the many years over which herbicide spraying occurred, it is unlikely that a single person would have participated every year in one or more of the activities described above. It is more likely that many different individuals were involved in these activities for various periods between 1948 and 1979.

4.5.2. Receptor characteristics

Predicting exposures required the use of many receptor characteristics. Table 4.27 summarizes the characteristics used to calculate exposure for each receptor group described above, with the exception of the mixer/loader, applicator, and flagman receptors. Occupational exposures (i.e., the mixer/loader, applicator, and flagman) do not depend on receptor characteristics or the amount of time spent at a given location but rather the amount of herbicide applied per day. The methods used to predict exposure for these receptors were those endorsed by Health Canada's Pest Management Regulatory Agency (PMRA) and used Pesticide Handlers Exposure Database (PHED) exposure parameters.

Table 4.27. *Summary of receptor parameters used to calculate exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Receptor parameter	Unit	Junior ranger	Recreational visitor	Resident/ bystander—toddler	Resident—adult	Hunter/ angler	First Nations person
Age range	yrs	12-19	20+	0.5-5	20+	20+	20+
Body weight	kg	59.7	70.7	16.5	70.7	70.7	70.7
Exposed surface area	cm^2	3858	4343	1745	4343	4343	4343
Incidental soil ingestion rate	g/d	0.05	0.05	0.2	0.05	0.05	0.05
Inhalation rate	m^3/d	15.6	16.6	8.3	16.6	16.6	16.6
Soil adherence factor—body	mg/cm^2/ event	0.07	0.07	0.2	0.07	0.07	0.07
Wild berry consumption rate	g/d	NA	5.2	1.2	5.2	NA	5.2
Local fish consumption rate[a]	g/d	NA	24.4	21.3	24.4	24.4	220
Wild game consumption rate[b]	g/d	NA	1.74	0.83	1.74	18	270
Fraction of fish from spray area[c]	NA	NA	NA	0.1	0.1	0.1	0.5
Fraction of wild game from spray area[d]	NA	NA	NA	0.5	0.5	0.5	0.25

NA = not applicable.
Health Canada 2010, MOE 2011, USEPA 2011.
[a] Local fish consumption rate: First Nations based on Health Canada (2010) data for total fish intake and assumption of 100% caught locally; hunter/angler estimates based on total fish intake (Health Canada 2010) and freshwater recreational catch fraction for lakes (US EPA 2011); toddler and adult assumed same as hunter/angler.
[b] Wild game consumption rate: First Nations based on Richardson (1997) data for wild game consumption estimates for Canadian native wild game eaters only; toddler and adult estimates based on total meat intake (Health Canada 2010) and wild game fraction of typical diet for US population from 1977 to 1978 (US EPA 2011); hunter/angler assumed 10x that of general population.
[c] Fraction of fish from spray area: assumed 10% for non-First Nations person and 50% for First Nations person.
[d] Fraction of wild game from spray area: assumed 50% for hunter/angler, toddler, and adult as non-native populations can only hunt limited quantities of deer and moose. First Nations assumed 25% as their higher dietary intake requires multiple catches from multiple areas.

4.5.3. Occupational exposure parameters

The panel estimated exposure for each occupational receptor using the U.S. EPA Occupational Pesticide Handler Unit Exposure Surrogate Reference Table (US EPA 2012) and PHED (US EPA 1998). The U.S. EPA table provides the most current data for assessing exposure and risk for occupational exposure handlers. In general, *unit exposures* are used as the basis for assessing occupational exposures to herbicides. The panel developed unique unit exposure values from available data sources for each receptor (mixer/loader, applicator, flagger), and these values are expressed as mass of pesticide active ingredient exposure per unit mass of active ingredient handled (e.g., µg/lb a.i.). Unit exposures are specific to the type of application equipment, formulation type, job function, and level of personal protective equipment used. These unit exposures, regardless of chemical identity, can be used to estimate exposure for any herbicide used in a particular spray scenario. The basic assumption is that herbicide handlers' exposure can be calculated generically based on the available empirical data for chemicals, as their exposure is primarily a function of the formulation type and the handling activities (e.g., packaging type, mixing/loading/application method, and clothing scenario) rather than chemical-specific properties.

The use of generic data that does not directly address a herbicide's chemical properties is appropriate. The experience in agriculture and forestry is that the formulation, the method of application, the label rate of application, the percent active ingredient, and the number of acres treated, but not the chemical properties of the product, determines the amount of dermal and inhalation exposure. This assumption allows exposure and

risk assessments to be conducted with many more observations than would be available from a single exposure study, thus increasing confidence in the assessment (US EPA 2007).

The Occupational Pesticide Handler Unit Exposure Surrogate Reference Table (US EPA 2012) is a quick reference guide that presents the current recommended unit exposures for standard occupational herbicide handler exposures. These values were derived from a number of sources, including PHED, the Outdoor Residential Exposure Task Force (ORETF), the Agricultural Handler Exposure Task Force (AHETF), and data from other available exposure monitoring studies.

Before the late 1990s, unit exposure estimates were generally derived from PHED. However, since then, both the ORETF and AHETF have produced pesticide handler exposure monitoring data that is now used by U.S. EPA in place of PHED. The U.S. EPA also recently evaluated other available occupational exposure monitoring studies and incorporated them into their occupational exposure estimates.

PHED is a generic database of exposure data for workers mixing/loading and applying pesticides under typical, actual field conditions, developed by a task force consisting of representatives from the U.S. EPA, Health Canada, the California Department of Pesticide Regulation, and member companies of the American Crop Protection Association (US EPA 2007). The database contains data for more than 2000 monitored exposure events. PHED is a compilation of passive dosimetry data for generic mixer/loader, flagmen, and applicator receptors, which allows generation of exposure estimates for specific spray scenarios. Exposure estimates are presented based on the best-fit measure of central tendency, i.e., summing the measure of central tendency for each body part that is most appropriate to the distribution of data for that body part. PHED appears to have been updated last in 1998 (US EPA 1998).

AHETF is a consortium of chemical companies established in December 2001 to address ongoing, product-specific data requirements. AHETF has developed a comprehensive database of information using a robust statistical design and improved analytical methods and is representative of current handling techniques that can be used to calculate exposure for major agricultural and non-agricultural handler scenarios. With joint input by Health Canada's PMRA and California's Department of Pesticide Regulation, U.S. EPA believes that AHETF data are superior and that using these data will result in more reliable and scientifically defensible exposure assessments. However, they continue to use data from PHED when more reliable data (e.g., AHETF data) are not available. Similarly, the panel used the more up-to-date AHETF data whenever possible, but in some cases found the AHETF database was insufficient and used data from PHED Version 1.1 instead.

4.5.3.1. Personal protective equipment

Occupational exposure assessments reflect that workers may use different levels of personal protective equipment. Using AHETF/PHED data for predicting exposure to the mixer/loader, flagmen, and applicator receptor groups allows for the use of exposure factors that depend on the level of personal protective equipment used during each activity.

Operational procedures provided to MTO and Ontario Hydro workers as far back as the 1950s and 1960s provide for use of appropriate (for the times) personal protective equipment (A0134563, A0134562, A0138491). For example, MTO (A0134563) indicated that employees should be issued specific items of protective and safety clothing (neoprene gloves and blue coveralls for weed spray crews) and that supervisors were responsible for ensuring its proper and intended use. Ontario Hydro (A0138491) advised that work should be conducted to minimize breathing in the spray mist and that direct contact between skin and herbicides should be avoided by using suitable clothing (coveralls, rubber boots, and gloves were recommended).

That said, the panel has to no way to know whether workers used personal protective equipment as advised. Thus, the panel considered three levels of personal protective equipment use:

1. The receptor wore an extra layer of clothing (coveralls over a single layer of clothing as above) with no respiratory protection and chemical-resistant gloves.
2. The receptor wore a single layer of clothing (long-sleeved shirt and long pants) with no respiratory protection and chemical-resistant gloves.
3. The receptor wore a single layer of clothing (long-sleeved shirt and long pants) with no respiratory protection and no chemical-resistant gloves.

These three levels represent low, central, and high exposure estimates to cover the range of potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic or likely estimate.

4.5.3.2. Herbicide formulation type

Predicting exposures during mixing and loading depends on the herbicide formulation type. PHED data is provided for these formulation types: dry flowable, granular, all liquids, wettable powder.

PHED data were selected according to the formulation types for 2,4,5-T and premix, which was assumed to be liquid in all cases.

4.5.3.3. Mixing and loading systems

Systems used to mix and load chemicals are generally defined as either open or closed. Open systems result in much higher rates of exposure than closed systems do. As with personal protective equipment, actual practices were not documented. Thus, the panel took the conservative route and used data based on use of open systems.

4.5.3.4. Application equipment

Based on a review of historical documentation, herbicides were sprayed using both aerial and ground-based methods. PHED data were available to describe dermal and inhalation exposure to an applicator using fixed-wing and rotary-wing aircraft and various ground application methods. The panel evaluated exposures and risks of the mixer/loader and applicator using aerial and ground spraying.

4.5.3.5. Unit exposure values

Table 4.28 provides the unit exposure (UE) values the panel used for this assessment.

Table 4.28. *Summary of unit exposure values used to calculate occupational exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Receptor	Dermal unit exposure (µg/lb a.i.)			Inhalation unit exposure (µg/lb a.i.)	Data source
	Double layer, gloves	Single layer, gloves	Single layer, no gloves	No respirator	
	Low[a]	Central	High		
Mixer/loader, liquids	29.1	37.6	220	0.219	AHETF[b]
Applicator, aerial, fixed wing (enclosed cockpit)	5	5	5	0.068	PHED[c]
Applicator, right-of-way sprayer	290	390	1300	3.9	PHED
Flagman, liquids	10.6	12	11	0.35	PHED
Mixer/loader/applicator, backpack sprayer	4120	8260	8260	2.58	U.S. EPA[d]

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[b] *AHETF = Agricultural Handler Exposure Task Force.*
[c] *PHED = Pesticide Handler Exposure Database.*
[d] *U.S. EPA = U. S. Environmental Protection Agency.*

4.5.3.6. Specific information on 2,4,5-T

Aerial spraying of 2,4,5-T

In a study designed to evaluate worker exposure during aerial application of herbicides, 2,4,5-T was supplied as 4 lbs acid-equivalent per gallon and formulated as propylene glycol butyl ether ester. All workers in crews were monitored for several methods during and after helicopter application. Helicopter operations used an application rate of 2 lbs/acre in 5 gals of water per acre. The exposure analysis focused on two separate helicopter crews made up of a pilot, a mixer, a supervisor, and two flagmen. Workers, for the most part, did not wear gloves or special protective clothing. The typical attire for spray crew members included long trousers, shirt (long or short sleeves), and field boots (Lavy et al. 1980).

Respiratory tract exposure to the 2,4,5-T ester was assessed via a portable air pump that drew a known volume of air to trap 2,4,5-T on a resin (XAD-2). Air was drawn through the resin at approximately 6 to 7 L of air/hour. Dermal exposure tests estimated quantity of 2,4,5-T likely to come into contact with bare skin. Six cellulose-backed gauze patches (10 x 10 cm) were attached to the clothing (chest, back, upper arms, and upper thighs) of each worker. The total amount of 2,4,5-T per patch times the total skin area exposed was assumed to constitute the total dermal exposure for the activity (Lavy et al. 1980).

Results

Only one of 10 helicopter application crew members (a flagman) showed any evidence of detectable levels of 2,4,5-T by use of air-monitoring devices, suggesting low potential for inhalation exposure. However, urine samples for each showed evidence of exposure to 2,4,5-T for mixers, suggesting that dermal exposure was a likely source.

Other members of the spray crew showed low but detectable levels of exposure. Mixers had the highest internal exposures, followed by helicopter pilots, supervisors, and flagmen. The apparently elevated exposure that one of the pilots experienced was attributed to the fact that he maintained the spray nozzles and helped change the spray boom before and after each flight (Lavy et al. 1980). Table 4.29 presents average levels of 2,4,5-T detected in the air and crew members' skin and urine samples following aerial application.

Table 4.29. *Average levels of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) detected in air (inhalation zone), skin (patches), and urine samples following helicopter application of herbicide.*[a]

Operation and duty	Air (breathing zone) µg/kg	Skin (mg/kg)	Urinary excretion[b] (mg/kg)
Pilot 1	ND[c]	0.110	0.005
Pilot 2	ND	ND	0.038
Mixer 1	ND	0.12	0.065
Mixer 2	ND	0.042	0.096
Supervisor 1	ND	0.024	0.004
Supervisor 2	ND	ND	0.003
Flagman 1	ND	ND	0.002
Flagman 2	ND	ND	0.001
Flagman 3	ND	ND	0.001
Flagman 4	1.03	ND	0.001

[a] *Adapted from Lavy et al. 1980.*
[b] *Total excreted over 6 days after exposure.*
[c] *ND = not detected.*

Conclusion

The experience with aerially applying herbicides to forests does not appear to contradict the information available in PHED. It is not clear whether using the exposure data from Lavy et al. (1980) could be usefully applied to the circumstances that prevailed in Ontario. As such, the generic PHED information was favoured.

4.5.4. Time activity factors

The level of exposure an individual may experience depends the amount of time spent at a specific area and activity. MOE (2011) provided generic exposure duration and frequency assumptions for different age groups and receptor activities, e.g., resident or worker (Table 4.30).

Table 4.30. *Time activity patterns for receptors used to calculate exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Time activity input parameter	Unit	Occupational	Non-occupational			
			Bystander	Resident	Recreational visitor	First Nations person
Number of hours spent per day	hour/day	10	1	24	4	24
Number of days per year[a]	day/year	180	1	275	14	275
Averaging time	days	365	365	365	365	365

[a] *Assumed average of 39 weeks of exposed soil per year (MOE 2011).*

4.6. Exposure pathways

To develop realistic exposure estimates, the panel needed details about where, when, and how various individuals might have come into contact with the chemicals of concern. Figure 4.5 provides an overall conceptual framework, which outlines the general exposure sources, the individuals of concern, and the various ways in which these receptor groups might have encountered the active ingredients.

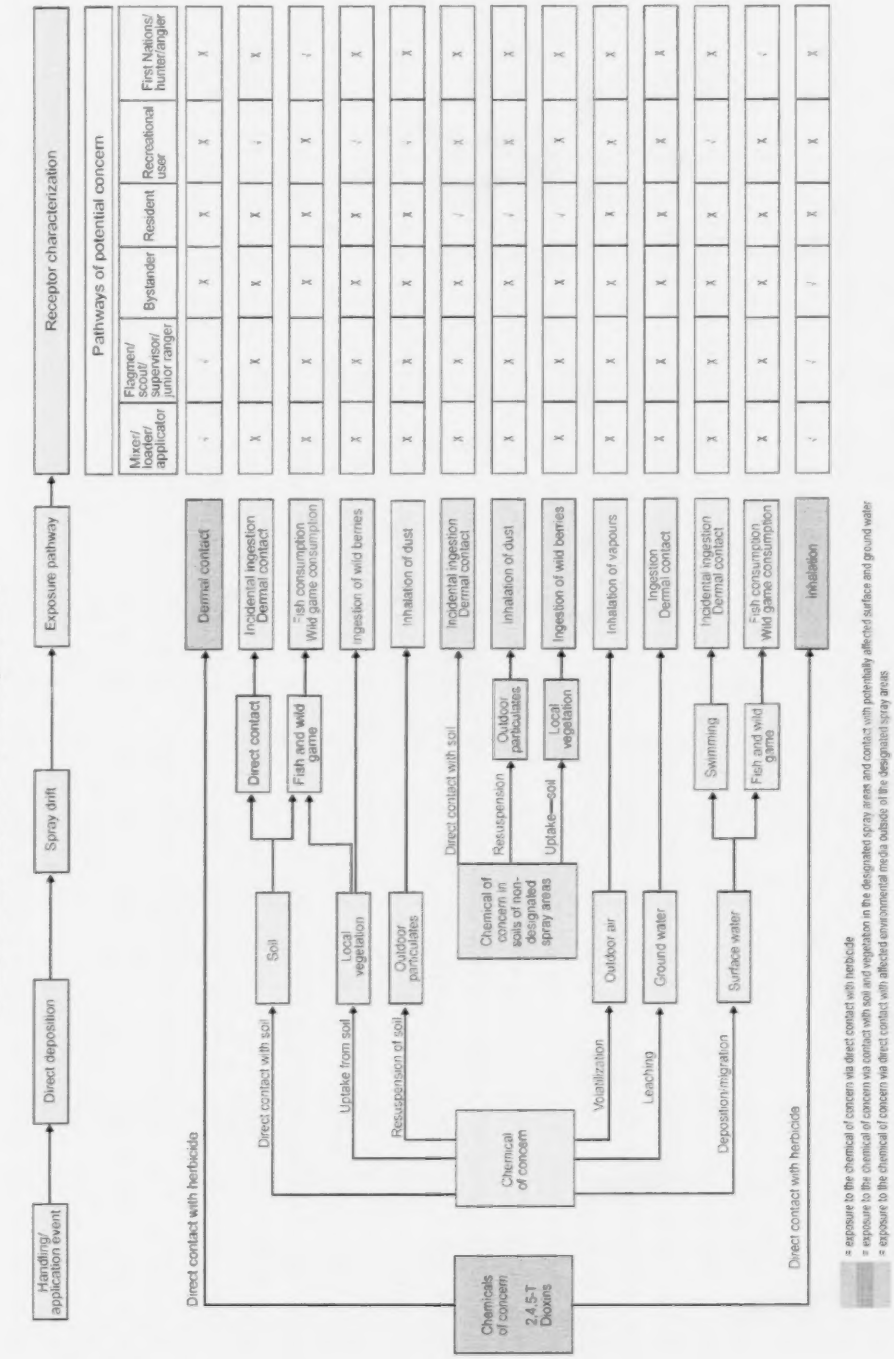


Figure 4.5. *Conceptual model for calculating exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

4.6.1. Major pathways

Exposures of individuals may be classified into two types: direct and indirect. Direct exposure results when an individual is in an area during spraying. Indirect exposure occurs when an individual comes into contact with environmental media that have been contaminated by spraying.

The panel considered these exposure pathways in the assessment:

- Direct dermal contact with herbicide
- Inhalation of herbicide
- Incidental ingestion of affected surface soil
- Direct dermal contact with soil
- Inhalation of re-suspended dust from soil
- Consumption of wild berries
- Consumption of wild game and fish

4.6.2. Minor pathways

Several potential exposure pathways were not considered because they could not be quantitatively assessed. These included transfer of herbicide residues from workers to other family members, including by tracking herbicides to the home; exposures related to burning of brush and/or forest fires; and consumption of ground and surface water.

The panel considered some pathways in a qualitative manner; these included transfer of herbicide residues from workers to other family members, including by tracking herbicides to the home; exposures related to burning of brush and/or forest fires; and consumption of ground and surface water.

Rather than providing meaningless and highly uncertain risk estimates, the panel considered these pathways qualitatively:

- **Ingestion of groundwater:** Ground water wells are located throughout Ontario. Receptors such as residents and workers likely consumed ground water at one time or another. Back extrapolation of potential ground water concentrations and exposures was deemed difficult and uncertain. MOE provided historical drinking water data that showed very limited levels of 2,4,5-T or TCDD in Ontario's water supply. Although this pathway was viable, the uncertainties inherent in any quantitative evaluation, coupled with anticipated low levels of exposure (due to the short persistence of 2,4,5-T and the low solubility of TCDD) resulted in the panel excluding this pathway from further consideration.
- **Ingestion of surface water:** Many surface water bodies are located throughout Ontario and may have been subject to direct spray and/or runoff. Various receptors—such as residents, recreational visitors, First Nations people, and workers—likely consumed water from these water bodies. MOE provided historical surface water data that showed very limited presence of 2,4,5-T or TCDD in Ontario's water supply. Although this pathway was viable, the uncertainties inherent in any quantitative evaluation, coupled with anticipated low levels of exposure (due to the short persistence of 2,4,5-T and the low solubility of TCDD) resulted in the panel excluding this pathway from further consideration.
- **Volatilization after application:** As it is unlikely that elevated concentrations of vapours will exist in ambient air for an extended period of time after application due to the short persistence of 2,4,5-T and the low volatility of TCDD, this pathway was not considered relevant. In addition, rapid volatilization of the active ingredient is likely not an intentional design characteristic given their intended use. Direct vapour inhalation while spraying and making direct dermal contact with soil were considered to be exposure pathways of most immediate concern, compared with indirect inhalation.

- **Take-home exposure pathway:** Limited information was available in the literature to address this issue. Experience with the chemicals of concerns evaluated in this assessment is limited; rather, reports regarding several pesticides are summarized below. However, some of these pesticides are more toxic than 2,4,5-T and TCDD and do not accurately represent the hazards of the latter. These examples are provided only to address concerns related to this pathway. Pesticide applicators may inadvertently carry hazardous materials home from work on their clothes, skin, hair, tools and in vehicles. Deaths and neurological effects in families of workers have been observed from pesticide contamination (NIOSH 1995). Some examples include a young girl who was poisoned due to contact with shoes contaminated with demeton (West 1959), and classic symptoms of organophosphorous pesticide exposure in spouses and children of greenhouse workers have been reported (Richter et al. 1992). However, in most situations, adverse effects have not been observed in family members of pesticide applicators, even in farm homes where increased pesticide concentrations have been measured in household dust (Curl et al. 2002, Fenske et al. 2002, Curwin et al. 2005, Coronado et al. 2006). For example, children living in homes with elevated concentrations of house dust contaminated with chlorpyrifos did not appear to have increased exposures, as measured through biological monitoring (Fenske et al. 2002).

  - Clothing

    Family members coming into contact with pesticides via touching contaminated work clothes may be a potential exposure pathway. Poor hygiene practices in a chemical plant that manufactured the pesticide kepone led to the contamination of workers' homes; workers' wives who reported that they washed their husbands' work clothes showed signs of kepone poisoning (Cannon et al. 1978).

    Incompletely removing pesticides from contaminated work clothes can result in increased exposure for pesticide applicators. Pesticide active ingredients or formulations that are more water-soluble are more effectively removed during laundering (Easley et al. 1983). For example, only 29 to 45% of the original 2,4-D ester contamination was removed from contaminated denim via laundering, whereas 99 to 100% of the water-soluble 2,4-D amine was removed (Easley et al. 1983). Studies of fabric contaminated with six classes of pesticides found that mean pesticide removal ranged from 65 to 100% (Braun et al. 1990, Nelson et al. 1992).

    With some pesticides, residue levels after laundering are toxic enough to kill certain insects (Laughlin et al. 1981) and to cause illness or contribute to mortality of adult males (Clifford and Nies 1969, Southwick et al. 1974). Given the high residue levels of pesticides that may remain on contaminated work clothes, this may be an exposure pathway for pesticide applicators. However, confounding factors such as water temperature, detergent, pre-wash treatment, water level, drying, pesticide formulation, time until laundering, repetitive washing, and laundry additives all affect the level of pesticide residues on clothing after washing (NIOSH 1995). In addition, the fabric treatment type (Laughlin and Gold 1989) and the pesticide formulation (Laughlin et al. 1981) affect a fabric's initial contamination level.

    Pesticide-contaminated clothing can in turn contaminate laundry equipment and clothing washed with it and/or in the next load. However, studies have shown that the transfer of pesticides from contaminated clothing to clean fabric washed in the same load ranged from 0 to 3.8% of the original residue level of the contaminated clothing (Easley et al. 1983, Laughlin and Gold 1989). When uncontaminated clothes were washed right after contaminated ones, the former had only <0.01% of the latter's contamination level (Laughlin and Gold 1989). Based on these data, pesticide transfer from contaminated work clothes to clean clothes during laundering does not appear to be a significant exposure pathway for family members.

  - Track-in exposure

    Evidence of the take-home exposure pathway exists (Curl et al. 2002, Fenske et al. 2002, Curwin et al. 2005, Coronado et al. 2006); however, the main transfer mechanism is unclear. Fenske et al. (2002) measured elevated chlorpyrifos concentrations in house dust in agricultural family homes located more

than a quarter mile from farmland. Gurnier et al. (2011) found that pesticide levels in house dust were related to proximity of use.

Fenske et al. (2002) found chlorpyrifos residues on work boots and children's hands for many of the agricultural families but none of the reference families. They detected very little chloropyrifos or parathion on steering wheels, which was assumed to be representative of worker skin and clothing contamination. In the homes of Iowa farms where pesticides had been applied within a week before sampling, researchers found that the entranceway, father's change area, and laundry room had the highest levels of the agricultural pesticides atrazine and metolachlor in dust (Curwin et al. 2005). These rooms are the most likely to have dirt tracked into or the farmer's clothes deposited in, suggesting farmers bring pesticide residues home on their clothing and shoes (Curwin et al. 2005). The organophosphate pesticide azinphos-methyl concentration in vehicle and house dust was highly correlated to metabolites measured in urine samples from adult and child relatives living on farms (Coronado et al. 2006). A correlation was measured between azinphos-methyl concentrations in vehicle and house dust, which is consistent with the assumption that farmers carry pesticides from the workplace to vehicles then homes via clothing, hats, and boots (Curl et al. 2002, Coronado et al. 2006).

A study by Nishioka et al. (2001) examined how 2,4-D was distributed in air and on surfaces of residences after lawn applications. 2,4-D was detected in indoor air and on all surfaces throughout the homes, with active dogs and homeowner-applicators being the most common sources. Applicators' shoes contributed significantly to the floor loading at 27%; however, active dogs accounted for >60% of that. Active children and dogs stirring up floor residues was the major source of 2,4-D in indoor air (Nishioka et al. 2001). Dogs' major contribution suggests the herbicide was tracked in after use near the home rather than a remote location such as a farm field. Curwin et al. (2005) showed that farms sprayed with pesticides had higher levels of pesticide in house dust than non-sprayed farms and non-farm homes, again showing a positive correlation between contaminant levels in the house and spraying proximity.

McCauley et al. (2001) examined whether residual levels of azinophos-methyl are linked to the number of agricultural workers in each household in migrant Latino farm worker communities in Oregon. They found that the median concentration of dust residues increased 170% for each additional farm worker living in the home.

- Conclusion about exposure of family members via the take-home pathway

  Compounds that are likely to cling to clothing, shoes, skin, or hair are potential take-home contaminants. Significant evidence of the take-home pathway for pesticides exists. Transportation of pesticides into homes appears to occur through tracking contaminated dirt into the home via work boots and contaminated clothing. However, it was not possible to quantify this exposure pathway due to the large number of variables and assumptions required. Therefore, exposure of family members to pesticides through the take-home exposure pathway was excluded as a quantitative pathway. It is reasonable to assume that family members' risks are much less than those of workers involved spray and/or forestry activities. The panel's inability to quantify exposure levels via this pathway should not be taken as evidence that such exposure is impossible.

- **Exposure after burning of treated vegetation:** Herbicides and insecticides have been used extensively during forestry operations for site preparation, release, and insect control. Prescribed or wild fires in forested areas treated with herbicides may result in the exposure of forest workers to volatilized herbicides or their combustion products.

  - Combustion products

    Forestry herbicides contain carbon, hydrogen, oxygen, nitrogen, chlorine, sulphur, and phosphorus. Almost all the carbon in any herbicide that undergoes chemical decomposition in a fire will become carbon dioxide and carbon monoxide (Dost 1982). Harmful combustion products could result during herbicide decomposition in a fire; for example, almost all of the chlorine from chlorine-containing

herbicides such as 2,4-D and 2,4,5-T will be converted to hydrogen chloride. In the laboratory, very small amounts of chlorine gas and phosgene were produced from 2,4-D under forced high pressure and high concentration conditions (Dost 1982). In addition, nitrogen-containing herbicides such as glyphosate, triclopyr, and hexazinone may produce ammonia and cyanide when burned, and phosphorus in glyphosate can form phosphorus pentoxide (in solution, phosphoric acid) and acetonitrile (Dost 1982).

Natural vegetation contains the same elements present in many common herbicides (carbon, hydrogen, oxygen, nitrogen, chlorine, sulphur, and phosphorus), but in different chemical arrangements and amounts (McMahon and Bush 1992). In a typical prescribed fire in herbicide-treated areas, grams to kilograms of herbicides are burned in the presence of tonnes of natural forest fuels. Therefore, even the trace levels of nitrogen and chlorine in forest fuels outweigh that from herbicides (McMahon and Bush 1992).

- Herbicide degradation

Studies conducted on herbicides and insecticides show that flaming combustion (>500 °C) thermally degrades most pesticides, whereas smouldering combustion (<500 °C) can volatilize significant amounts of some pesticides. In most wood fires, both processes occur, but one usually dominates, depending on the fuel and environmental conditions (McMahon et al. 1985). Bush et al. (1998) showed that rapid, flaming combustion is associated with wildfires. Thus, during wildfires most pesticides will be thermally degraded.

In a forest fuel combustion study, wood treated with five herbicides and two insecticides was burned via controlled combustion (McMahon et al. 1985). When wood burned under rapid combustion (flaming), over 95% of pesticides decomposed. During smouldering combustion, varying amounts of pesticide residues were recovered from the smoke. Slow heating or smouldering conditions will likely release relatively stable compounds such as lindane and dicamba, as well as compounds with significant vapour pressures (i.e., hexachlorobenzene). For example, lindane, dicamba, and 2,4-D were recovered intact in the smoke at 43, 92, and 92% of their applied amounts, respectively, whereas chlorpyrifos, picloram, and hexazinone were extensively decomposed (>75%) (McMahon et al. 1985).

Bush et al. (1998) found similar results during laboratory combustion of firewood treated with phenoxy and pyridine herbicides. During slow combustion (<500 °C), relatively stable compounds such as 2,4-D, dicamba, and dichlorprop were released in significant amounts (88.9, 91.5 and 100%, respectively). Carryover during slow heating depended on the herbicide's physical properties. During flaming combustion (500 °C), recovery of the herbicide active ingredient ranged from 0 to 32% for picloram and dicamba, respectively (Bush et al. 1998). Active flaming combustion (800 to 1000 °C) were predicted to result in >95% decomposition of the phenoxy and pyridine herbicides (2,4-D, dicamba, dichlorprop, picloram, triclopyr) examined (Bush et al. 1998).

Laboratory studies show that at least 90% of parent herbicides decompose when herbicides are subjected to combustion conditions found in prescribed burning (McMahon et al. 1985, McMahon and Bush 1986).

- Forestry worker exposure

McMahon and Bush (1992) attempted to measure herbicide concentrations in smoke from prescribed burning operations ("brown and burn") of forests 30 to 169 days after herbicide treatment with the active ingredients imazapyr, triclopyr, hexazinone, and picloram. No herbicide residues were detected (sensitivity 0.1 to 4 $\mu g/m^3$) in 140 smoke samples from 14 fires (McMahon and Bush 1992).

USDA (1989a,b,c) conducted a modelling assessment that showed that airborne herbicides posed an insignificant risk to forest workers even if the fire occurs right after spraying. To be conservative, this model assumed no herbicide decomposition.

Forestry workers' exposure to herbicides during prescribed burning of pesticide-treated wood has been modelled before (Dost 1982, USDA 1989a,b,c). However, these estimates have a high level of uncertainty, such as:

- Time between pesticide application and prescribed burning
- Fraction of pesticide/contaminant carryover in smoke condensate
- Distribution of combustion products
- Exposure frequency, time, and duration

Due to the high level of uncertainty and the assumptions associated with these exposure estimates, it is not possible to quantitatively evaluate pesticide exposure from inhaling smoke. Based on the available information, smoke inhalation is likely not a significant pathway.

4.7. Additional exposure variables

4.7.1. Exposure duration

The frequency and duration of herbicide applications varied significantly among different scenarios so not all scenarios were treated the same way. In some areas, herbicides were applied only once (MNR); in others, they were applied annually or semi-annually (MTO) or on a five- to six-year cycle (Ontario Hydro). Also, some receptors (occupational) could have been exposed repeatedly, with others exposed only intermittently (recreational visitors) or once (bystanders). As a result, the panel considered two possible exposure durations:

- Short term (acute):
  - ranged from one to seven days
  - stemmed from a single herbicide spray
  - was applied to the bystander
- Long term (chronic):
  - was greater than six months (as per U.S. EPA standard)
  - was assumed to be approximately six months to entire lifetime
  - was applied to those involved in regular, repeated herbicide spraying/exposure

The panel calculated chronic occupational exposures since workers sprayed for the summer season and often were involved for multiple years. All non-occupational receptors (except for bystander—preschool child) were also evaluated under a chronic exposure scenario since exposures may have occurred over a long period or repeatedly. The averaging time used in all chronic exposure estimates was 180 days (or six months). This value was considered the minimum chronic exposure duration, and in most areas of Ontario herbicide spraying occurred between May and October. Also, due to the persistent nature of TCDD, it would likely remain in the environment long after 2,4,5-T was sprayed, thereby leading to chronic exposure.

4.7.2. Population proximity

Herbicide spraying occurred in remote areas as well as close to more populated areas. As such, two population proximities were considered in the assessment:

- Remote (associated with aerial applications)
- Residential (associated with ground applications)

MNR used herbicides for forestry purposes, generally in remote areas, with most spraying done aerially. MTO used ground-based methods to apply herbicides adjacent to roadways in both urban and rural areas. Ontario Hydro applied along transmission corridors in both remote and populated areas via both aerial- and ground-based methods.

4.7.3. Relative absorption factors (RAFs)

The bioavailability of a chemical defines the fraction of the chemical that is available to produce toxic effects at a particular site of action (i.e., target organ). Thus, bioavailability can represent the difference between the administered and the absorbed dose. Since most toxicological reference values are based on administered doses, it is generally not appropriate to consider absolute bioavailability (i.e., proportion of the administered dose that reaches the systemic circulation) during exposure assessments. A better measure may be relative bioavailability or absorption, which can be determined by comparing the extent of absorption among several routes of exposure, forms of the same chemical, or administration vehicles (food, soil, water). Absorption may differ according to whether the dose was received through the skin, ingested, or inhaled. Absorption may also differ depending on how the chemical is delivered (a solvent vehicle, water, soil, food, etc.)

When a toxicological reference value is not available for the exposure route of concern, it is often necessary to consider route-to-route extrapolation. For example, it is common practice to evaluate the risks that dermal absorption of a chemical poses using a toxicological reference value established via the oral route. The dose absorbed through the skin is scaled to the equivalent oral dose by correcting for differences in route-specific bioavailabilities (i.e., the proportion absorbed via the dermal vs. oral route). When route-to-route extrapolation is not necessary (i.e., oral and inhalation exposures), relative absorption is not typically adjusted.

The relative absorption difference between the oral and dermal exposure routes is often expressed as a relative absorption factor (RAF_{dermal}), and is applied to dermal exposure estimates to adjust them before comparing with oral toxicological reference values when route-to-route extrapolation is necessary. This factor is calculated as follows:

$$RAF_{dermal} = \frac{AF_{dermal}}{AF_{oral}} \qquad \text{(Equation 4.13)}$$

The oral bioavailability of a chemical is typically determined from absorption or excretion studies and is generally assumed to be 100% minus the percent excreted in the feces. When only the fraction of chemical in the urine is reported, this fraction is selected as the minimum oral bioavailability, with the maximum being 100%. Although it is unlikely that 100% of a compound is excreted in the urine (since some may be deposited in tissue or expired), it is equally unlikely that 100% will be absorbed (since some may be excreted in the feces). If no data indicates otherwise, the oral bioavailability of a compound in the toxicological study used to derive an oral toxicological reference value is assumed to be approximately 100%. The panel assumed that all herbicides were 100% bioavailable during the oral toxicological study; therefore, they used an oral absorption factor (AF_{oral}) of 1.0 to derive RAF_{dermal} values when an oral toxicological reference value was used to characterize risks.

Receptors were assumed to come into direct contact with herbicide mixtures while preparing mixtures or during application. The panel used a method described by the Syracuse Research Corporation (SERA 2007) to predict what

proportion of the applied dermal dose the skin may absorb. According to SERA (2007), dermal absorption rates (or k_a values expressed as amount per unit time or as reciprocal time) and dermal penetration rates (or k_p values expressed as cm/hour) are commonly required for dermal exposure assessments. In addition, dermal absorption rates are used to estimate the absorbed dose associated with dermal deposition, and dermal absorption rates (k_a values) express the amount (zero-order) or proportion (first-order) of chemical absorbed into the body per unit time. Based on work conducted by Durkin et al. (1995), SERA (2007) concluded that first-order dermal absorption rates (k_a values) rather than penetration rates (k_p) should be used when describing exposures involving the deposition of a chemical onto the skin surface. The use of k_p values is more applicable to describing a situation where the skin is immersed in an aqueous solution. SERA (2007) determined that dermal first-order absorption rate coefficients were best described using both molecular weight and the logarithm of the octanol-water partitioning coefficient (K_{ow}) as follows:

$$Log_{10}k_{a(first\ order)} = 0.233 Log_{10}K_{ow} - 0.00566MW - 1.49 \qquad \text{(Equation 4.14)}$$

Where:

k_a = Chemical-specific first-order absorption dermal rate coefficient (hr^{-1})
K_{ow} = Chemical-specific octanol-water partitioning coefficient (unitless)
MW = Chemical-specific molecular weight (g/mol)

The methods provided by SERA (2007) were used to derive first-order dermal absorption rates (k_a) for 2,4,5-T and TCDD (Table 4.31).

Table 4.31. *First-order dermal absorption rates for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Chemical	Molecular weight	$Log_{10}K_{ow}$	$Log_{10}K_a$	k_a
2,4,5-T	255.49	3.13	-2.19	0.0064
TCDD	322	6.8	-1.71	0.020

As indicated, first-order dermal rate constants are time dependent; therefore, the amount of time a liquid is in contact with exposed skin must be provided. The following equation provides the method used to derive the proportion of chemical absorbed per unit time.

$$P = 1 - e^{-k_a t} \qquad \text{(Equation 4.15)}$$

Where:

P = proportion of chemical absorbed through the skin in time *t*
k_a = first-order absorption dermal rate coefficient
t = amount of time exposed skin is in contact with contaminant (10 hours for worker, one hour for bystander, four hours for recreational visitor, 24 hours for resident)

Table 4.32 presents the proportion of TCDD and 2,4,5-T absorbed through the skin.

Table 4.32. *Proportion of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) dermally absorbed per exposure activities.*

Chemical	Proportion absorbed through skin			
	Worker	Bystander	Recreational visitor	Resident
2,4,5-T	0.062	0.0064	0.025	0.143
TCDD	0.18	0.019	0.075	0.375

Table 4.33 summarizes the absorption rates and bioaccessibility of 2,4,5-T and TCDD through other routes of exposure.

Table 4.33. *Absorption rates and bioaccessibilities of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Parameter	Value	Reference
2,4,5-T soil bioaccessibility factor	1	Assumed
2,4,5-T oral absorption factor	1	Assumed
2,4,5-T inhalation absorption factor	1	Assumed
TCDD soil bioaccessibility factor	0.34	Ruby et al. 2002
TCDD oral absorption factor	1	Assumed (MOE 2011)
TCDD inhalation absorption factor	1	Assumed

4.8. Detailed exposure methods

The following sections describe the methods the panel used to calculate exposure to receptors within each receptor group.

4.8.1. Occupational receptors involved in spraying herbicides

To evaluate occupational receptors, the panel used PHED unit exposure values (UEs) to predict inhalation and dermal exposure experienced by herbicide workers (mixer/loader, applicator, flagman) during routine activities. It was assumed that all herbicides were in liquid form and that mixing and loading were done using open systems. The PHED dermal UEs, which are an expression of the amount of chemical the receptor absorbed dermally based on the amount of chemical handled, depend on the use of personal protective equipment. The PHED approach to predicting individuals' exposure relies on the mass of material handled or applied per day (rather than the mass applied per year, as described in Section 4.3); annual herbicide use rates were converted to daily use rates. The USDA (1989a,b,c) showed that the maximum number of hectares that could be treated with herbicides in one day via aerial and ground (boom-spray truck) spraying was 320 ha and 67 ha, respectively.

Table 4.34 provides a comparison of the maximum spray distance (USDA 1989a,b,c) and the theoretical spray distance, which is based on the actual distance sprayed per year (Table 4.10), the theoretical number of spray teams (36 for MTO, based on MTO spray records and 12 for Ontario Hydro, assumed based on their covering approximately one third the distance of MTO annually), and the length of the spray season (assumed May to October based on MTO spray records). For MNR, spraying occurred as discrete events over an extended period of time. The panel assumed a single spray team, consisting of an aerial crew, a team of mixers/loaders and flagmen, and a team of backpack sprayers (12-person team).

Table 4.34. *Maximum spray distances for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Measure considered	Aerial linear miles Ontario Hydro	Ground linear miles Ministry of Transportation	Ground linear miles Ontario Hydro
Maximum distance sprayed per day	65.9	45.6	13.8
Theoretical distance sprayed per day	<3	<85	<25

Exposures of the mixer/loader, applicator, and flagman to 2,4,5-T and TCDD were assumed to occur during routine mixing and loading activities via inhalation of vapours and dermal absorption. Since the exposure studies used to develop the PHED unit exposure values are passive dosimetry studies, in which the amount of pesticide deposited on clothing and skin and in the breathing zone of the worker is measured, these values represent exposure as a result of routine activities as well as common accidental exposure events. PHED provides generic passive dosimetry data for generic mixer/loader/applicator exposure scenarios specific to the formulation type, mixing and loading systems, application equipment, and level of personal protective equipment. PHED unit exposure values (UEs) were used to predict exposures under this generic scenario. The panel assumed that all herbicide active ingredients were in liquid form and that mixing and loading were done using open systems.

With the PHED approach to predicting exposures of individuals to herbicide mixtures, body weight (70.7 kg as per Health Canada 2010 and MOE 2011) was the only receptor-specific input parameter required. In other words, the PHED approach expresses dermal exposure (for routine activities) as a function of the mass of active ingredient or adjuvant handled per day (kg/day) and the dermal unit exposure value (mg/lbs). A body weight of 70.7 kg was used for all exposure levels (i.e., minimum, average, and maximum) for all scenarios except the junior ranger, for which a body weight of 59.7 kg was used (Health Canada 2010, MOE 2011).

Dermal exposures were adjusted by the chemical-specific dermal absorption factor.

Using the PHED UEs presented in Table 4.28 and the mass of active ingredient handled per day, the panel estimated dermal and inhalation exposures using equations 4.16 and 4.17, respectively.

$$Dose_{dermal} = \frac{AR \times SpA_{daily} \times UE_{dermal} \times AF_{dermal} \times ED}{BW \times DY} \qquad \text{(Equation 4.16)}$$

where:

- $Dose_{dermal}$ = absorbed dermal dose from routine activities (µg/kg BW/day)
- AR = application rate (lbs/acres; scenario and year specific)
- SpA_{daily} = daily spray area (acres/day; scenario and year specific)
- ED = exposure duration (180 days/year; assumed)
- UE_{dermal} = dermal unit exposure (µg/lbs; receptor specific)
- AF_{dermal} = dermal absorption factor
- DY = days per year (365 days/year)
- BW = body weight (kg)

And,

$$Dose_{inhalation} = \frac{AR \times SpA_{daily} \times UE_{inhalation} \times AF_{inhalation} \times ED}{BW \times DY}$$

(Equation 4.17)

where:

$Dose_{inhalation}$	=	absorbed inhalation dose from routine activities (µg/kg BW/day)
AR	=	application rate (lbs/acres; scenario and year specific)
SpA_{daily}	=	daily spray area (acres/day; scenario and year specific)
ED	=	exposure duration (180 days/year; assumed)
$UE_{inhalation}$	=	inhalation unit exposure (µg/lbs; receptor specific)
$AF_{inhalation}$	=	inhalation absorption factor (unitless)
DY	=	days per year (365 days/year)
BW	=	body weight (kg)

4.8.2. Occupational exposure estimates

The following sections provide the exposure estimates for individuals involved in spray operations. Results are provided for three spray scenarios (MTO, Ontario Hydro, MNR), all years spray activities occurred (scenario specific), all relevant occupational receptors (mixer/loader and applicator for both aerial and ground applications), three levels of personal protective equipment use, and varying estimates of contaminant levels (tables 4.35 to 4.40).

Table 4.35. *Estimates of Ministry of Transportation occupational exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Year	PHED[a] data—mixer/loader total (µg/kg/day)						PHED data—right-of-way applicator total (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[b]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1953	4.04E-02	5.09E-02	2.77E-01	5.35E-09	1.78E-06	3.67E-05[c]	4.35E-01	5.60E-01	1.69E+00	5.48E-08	1.89E-05	2.19E-04
1954	4.05E-02	5.11E-02	2.78E-01	5.37E-09	1.79E-06	3.68E-05	4.37E-01	5.62E-01	1.69E+00	5.50E-08	1.90E-05	2.20E-04
1955	4.15E-02	5.23E-02	2.84E-01	5.49E-09	1.83E-06	3.77E-05	4.47E-01	5.75E-01	1.73E+00	5.63E-08	1.94E-05	2.25E-04
1956	4.11E-02	5.19E-02	2.82E-01	5.45E-09	1.82E-06	3.74E-05	4.44E-01	5.71E-01	1.72E+00	5.59E-08	1.93E-05	2.23E-04
1957	4.26E-02	5.37E-02	2.92E-01	5.64E-09	1.88E-06	3.87E-05	4.59E-01	5.91E-01	1.78E+00	5.79E-08	1.99E-05	2.31E-04
1958	3.12E-02	3.93E-02	2.14E-01	4.14E-09	1.38E-06	2.84E-05	3.36E-01	4.33E-01	1.30E+00	4.24E-08	1.46E-05	1.69E-04
1959	3.42E-02	4.31E-02	2.35E-01	4.54E-09	1.51E-06	3.11E-05	3.69E-01	4.75E-01	1.43E+00	4.65E-08	1.60E-05	1.86E-04
1960	3.82E-02	4.82E-02	2.62E-01	5.06E-09	1.68E-06	3.47E-05	4.12E-01	5.30E-01	1.60E+00	5.19E-08	1.79E-05	2.07E-04
1961	3.95E-02	4.98E-02	2.70E-01	5.23E-09	1.74E-06	3.59E-05	4.25E-01	5.48E-01	1.65E+00	5.36E-08	1.85E-05	2.14E-04
1962	4.51E-02	5.69E-02	3.09E-01	5.98E-09	1.99E-06	4.10E-05	4.86E-01	6.26E-01	1.88E+00	6.13E-08	2.11E-05	2.45E-04
1963	5.89E-02	7.43E-02	4.04E-01	7.81E-09	2.60E-06	5.35E-05	6.35E-01	8.18E-01	2.46E+00	8.01E-08	2.76E-05	3.20E-04
1964	6.29E-02	7.93E-02	4.31E-01	8.33E-09	2.77E-06	5.71E-05	6.78E-01	8.73E-01	2.63E+00	8.54E-08	2.94E-05	3.41E-04
1965	5.58E-02	7.03E-02	3.82E-01	7.39E-09	2.46E-06	5.07E-05	6.01E-01	7.74E-01	2.33E+00	7.58E-08	2.61E-05	3.03E-04
1966	6.33E-02	7.98E-02	4.34E-01	8.39E-09	2.79E-06	5.75E-05	6.82E-01	8.79E-01	2.65E+00	8.60E-08	2.96E-05	3.44E-04
1967	8.07E-02	1.02E-01	5.53E-01	1.07E-08	3.56E-06	7.33E-05	8.70E-01	1.12E+00	3.37E+00	1.10E-07	3.78E-05	4.38E-04
1968	7.82E-02	9.85E-02	5.36E-01	1.04E-08	3.45E-06	7.10E-05	8.42E-01	1.08E+00	3.27E+00	1.06E-07	3.66E-05	4.24E-04
1969	7.81E-02	9.85E-02	5.35E-01	1.04E-08	3.45E-06	7.10E-05	8.42E-01	1.08E+00	3.26E+00	1.06E-07	3.66E-05	4.24E-04
1970	8.40E-02	1.06E-01	5.76E-01	1.11E-08	3.71E-06	7.63E-05	9.06E-01	1.17E+00	3.51E+00	1.14E-07	3.93E-05	4.56E-04
1971	7.15E-02	9.01E-02	4.90E-01	9.47E-08	1.21E-07	6.91E-07	7.71E-01	9.92E-01	2.99E+00	9.71E-07	1.29E-06	4.13E-06
1972	7.98E-02	1.01E-01	5.47E-01	1.06E-07	1.35E-07	7.71E-07	8.60E-01	1.11E+00	3.33E+00	1.08E-06	1.44E-06	4.61E-06
1973	7.98E-02	1.01E-01	5.47E-01	1.06E-07	1.35E-07	7.71E-07	8.60E-01	1.11E+00	3.33E+00	1.08E-06	1.44E-06	4.61E-06

[a] *PHED = Pesticide Handler Exposure Database.*
[b] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[c] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 4.36. *Estimates of Ontario Hydro occupational exposure (aerial) to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Year	PHED[a] data—applicator—aerial, fixed wing (µg/kg/day)						PHED data—flagman—aerial, fixed wing (µg/kg/day)						PHED data—mixer/loader—aerial, fixed wing (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[b]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1953	6.25E-03	6.25E-03	6.25E-03	6.58E-11	1.71E-08	6.18E-08	1.66E-02	1.81E-02	1.70E-02	1.54E-10	4.44E-08	1.49E-07	3.34E-02	4.22E-02	2.29E-01	3.71E-10	1.23E-07	2.54E-07
1954	1.53E-02	1.53E-02	1.53E-02	1.60E-10	4.16E-08	1.50E-07	4.06E-02	4.41E-02	4.16E-02	3.74E-10	1.08E-07	3.63E-07	8.17E-02	1.03E-01	5.60E-01	9.02E-10	3.00E-07	6.18E-07
1955	2.95E-03	2.95E-03	2.95E-03	3.10E-11	8.06E-09	2.92E-08	7.84E-03	8.52E-03	8.04E-03	7.24E-11	2.09E-08	7.03E-08	1.58E-02	1.99E-02	1.08E-01	1.75E-10	5.82E-08	1.20E-07
1956	9.10E-03	9.10E-03	9.10E-03	9.55E-11	2.48E-08	8.98E-08	2.42E-02	2.63E-02	2.48E-02	2.23E-10	6.45E-08	2.16E-07	4.88E-02	6.15E-02	3.34E-01	5.38E-10	1.79E-07	3.69E-07
1957	5.03E-02	5.03E-02	5.03E-02	5.27E-10	1.37E-07	4.96E-07	1.34E-01	1.45E-01	1.37E-01	1.23E-09	3.56E-07	1.19E-06	2.69E-01	3.39E-01	1.84E+00	2.97E-09	9.89E-07	2.04E-06
1958	5.69E-02	5.69E-02	5.69E-02	5.98E-10	1.55E-07	5.62E-07	1.52E-01	1.65E-01	1.55E-01	1.40E-09	4.04E-07	1.35E-06	3.05E-01	3.84E-01	2.09E+00	3.37E-09	1.12E-06	2.31E-06
1959	4.83E-02	4.83E-02	4.83E-02	5.06E-10	1.32E-07	4.76E-07	1.29E-01	1.40E-01	1.32E-01	1.18E-09	3.42E-07	1.15E-06	2.59E-01	3.26E-01	1.77E+00	2.85E-09	9.49E-07	1.96E-06
1960	6.18E-02	6.18E-02	6.18E-02	6.49E-10	1.69E-07	6.10E-07	1.65E-01	1.79E-01	1.69E-01	1.52E-09	4.38E-07	1.47E-06	3.31E-01	4.17E-01	2.27E+00	3.66E-09	1.22E-06	2.51E-06
1961	5.07E-02	5.07E-02	5.07E-02	5.32E-10	1.38E-07	5.00E-07	1.35E-01	1.47E-01	1.38E-01	1.24E-09	3.59E-07	1.21E-06	2.72E-01	3.42E-01	1.86E+00	3.00E-09	9.98E-07	2.06E-06
1962	4.11E-02	4.11E-02	4.11E-02	4.32E-10	1.12E-07	4.06E-07	1.10E-01	1.19E-01	1.12E-01	1.01E-09	2.91E-07	9.78E-07	2.20E-01	2.78E-01	1.51E+00	2.43E-09	8.10E-07	1.67E-06
1963	3.29E-02	3.29E-02	3.29E-02	3.45E-10	8.97E-08	3.24E-07	8.76E-02	9.52E-02	8.98E-02	8.05E-10	2.33E-07	7.81E-07	1.76E-01	2.22E-01	1.21E+00	1.94E-09	6.47E-07	1.33E-06
1964	3.63E-02	3.63E-02	3.63E-02	3.81E-10	9.90E-08	3.58E-07	9.66E-02	1.05E-01	9.90E-02	8.90E-10	2.57E-07	8.63E-07	1.94E-01	2.45E-01	1.33E+00	2.15E-09	7.14E-07	1.47E-06
1965	4.58E-02	4.58E-02	4.58E-02	4.80E-10	1.25E-07	4.51E-07	1.22E-01	1.32E-01	1.25E-01	1.12E-09	3.24E-07	1.09E-06	2.45E-01	3.09E-01	1.68E+00	2.71E-09	9.01E-07	1.85E-06
1966	4.24E-02	4.24E-02	4.24E-02	4.44E-10	1.15E-07	4.18E-07	1.13E-01	1.23E-01	1.16E-01	1.04E-09	3.00E-07	1.01E-06	2.27E-01	2.86E-01	1.55E+00	2.50E-09	8.33E-07	1.72E-06
1967	5.91E-02	5.91E-02	5.91E-02	6.20E-10	1.61E-07	5.83E-07	1.57E-01	1.71E-01	1.61E-01	1.45E-09	4.19E-07	1.41E-06	3.17E-01	3.99E-01	2.17E+00	3.50E-09	1.16E-06	2.40E-06
1968	5.91E-02	5.91E-02	5.91E-02	6.20E-10	1.61E-07	5.83E-07	1.57E-01	1.71E-01	1.61E-01	1.45E-09	4.19E-07	1.41E-06	3.17E-01	3.99E-01	2.17E+00	3.50E-09	1.16E-06	2.40E-06
1969	5.91E-02	5.91E-02	5.91E-02	6.20E-10	1.61E-07	5.83E-07	1.57E-01	1.71E-01	1.61E-01	1.45E-09	4.19E-07	1.41E-06	3.17E-01	3.99E-01	2.17E+00	3.50E-09	1.16E-06	2.40E-06
1970	5.91E-02	5.91E-02	5.91E-02	6.20E-10	1.61E-07	5.83E-07	1.57E-01	1.71E-01	1.61E-01	1.45E-09	4.19E-07	1.41E-06	3.17E-01	3.99E-01	2.17E+00	3.50E-09	1.16E-06	2.40E-06
1971	5.91E-02	5.91E-02	5.91E-02	6.20E-09	6.20E-09	6.20E-09	1.57E-01	1.71E-01	1.61E-01	1.45E-08	1.61E-08	1.49E-08	3.17E-01	3.99E-01	2.17E+00	3.50E-08	4.48E-08	2.55E-07
1972	5.91E-02	5.91E-02	5.91E-02	6.20E-09	6.20E-09	6.20E-09	1.57E-01	1.71E-01	1.61E-01	1.45E-08	1.61E-08	1.49E-08	3.17E-01	3.99E-01	2.17E+00	3.50E-08	4.48E-08	2.55E-07
1973	5.91E-02	5.91E-02	5.91E-02	6.20E-09	6.20E-09	6.20E-09	1.57E-01	1.71E-01	1.61E-01	1.45E-08	1.61E-08	1.49E-08	3.17E-01	3.99E-01	2.17E+00	3.50E-08	4.48E-08	2.55E-07
1974	5.91E-02	5.91E-02	5.91E-02	1.24E-09	1.24E-09	1.24E-09	1.57E-01	1.71E-01	1.61E-01	2.90E-09	3.22E-09	2.99E-09	3.17E-01	3.99E-01	2.17E+00	6.99E-09	8.95E-09	5.10E-08
1975	5.91E-02	5.91E-02	5.91E-02	1.24E-09	1.24E-09	1.24E-09	1.57E-01	1.71E-01	1.61E-01	2.90E-09	3.22E-09	2.99E-09	3.17E-01	3.99E-01	2.17E+00	6.99E-09	8.95E-09	5.10E-08
1976	5.91E-02	5.91E-02	5.91E-02	1.24E-09	1.24E-09	1.24E-09	1.57E-01	1.71E-01	1.61E-01	2.90E-09	3.22E-09	2.99E-09	3.17E-01	3.99E-01	2.17E+00	6.99E-09	8.95E-09	5.10E-08
1977	5.91E-02	5.91E-02	5.91E-02	1.24E-09	1.24E-09	1.24E-09	1.57E-01	1.71E-01	1.61E-01	2.90E-09	3.22E-09	2.99E-09	3.17E-01	3.99E-01	2.17E+00	6.99E-09	8.95E-09	5.10E-08
1978	5.91E-02	5.91E-02	5.91E-02	1.24E-09	1.24E-09	1.24E-09	1.57E-01	1.71E-01	1.61E-01	2.90E-09	3.22E-09	2.99E-09	3.17E-01	3.99E-01	2.17E+00	6.99E-09	8.95E-09	5.10E-08
1979	5.91E-02	5.91E-02	5.91E-02	1.24E-09	1.24E-09	1.24E-09	1.57E-01	1.71E-01	1.61E-01	2.90E-09	3.22E-09	2.99E-09	3.17E-01	3.99E-01	2.17E+00	6.99E-09	8.95E-09	5.10E-08

[a] *PHED = Pesticide Handler Exposure Database.*

[b] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[c] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 4.37. *Estimates of Ontario Hydro occupational exposure (ground) to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Year	PHED[b] data—mixer/loader (ground only) total (µg/kg/day)						PHED data—right-of-way applicator total (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[c]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1948	2.90E-04	3.66E-04	1.99E-03	4.19E-11	1.40E-08	2.88E-07	3.13E-03	4.03E-03	1.21E-02	4.30E-10	1.48E-07	1.72E-06
1949	1.09E-03	1.37E-03	7.44E-03	1.47E-10	4.89E-08	1.01E-06	1.17E-02	1.51E-02	4.54E-02	1.51E-09	5.18E-07	6.01E-06[d]
1950												
1951	1.71E-02	2.16E-02	1.17E-01	2.27E-09	7.56E-07	1.56E-05	1.85E-01	2.38E-01	7.16E-01	2.33E-08	8.02E-06	9.31E-05
1952	3.50E-02	4.42E-02	2.40E-01	4.64E-09	1.55E-06	3.18E-05	3.78E-01	4.86E-01	1.46E+00	4.76E-08	1.64E-05	1.90E-04
1953	5.70E-02	7.19E-02	3.91E-01	7.93E-09	2.64E-06	5.44E-05	6.15E-01	7.92E-01	2.38E+00	8.13E-08	2.80E-05	3.25E-04
1954	5.90E-02	7.44E-02	4.05E-01	8.73E-09	2.90E-06	5.98E-05	6.36E-01	8.19E-01	2.47E+00	8.94E-08	3.08E-05	3.57E-04
1955	8.72E-02	1.10E-01	5.98E-01	1.17E-08	3.91E-06	8.04E-05	9.40E-01	1.21E+00	3.64E+00	1.20E-07	4.14E-05	4.81E-04
1956	8.40E-02	1.06E-01	5.76E-01	1.17E-08	3.89E-06	8.00E-05	9.06E-01	1.17E+00	3.51E+00	1.20E-07	4.12E-05	4.78E-04
1957	1.40E-01	1.77E-01	9.63E-01	2.16E-08	7.18E-06	1.48E-04	1.51E+00	1.95E+00	5.87E+00	2.21E-07	7.62E-05	8.84E-04
1958	1.52E-01	1.92E-01	1.04E+00	2.35E-08	7.83E-06	1.61E-04	1.64E+00	2.11E+00	6.36E+00	2.41E-07	8.31E-05	9.64E-04
1959	1.45E-01	1.83E-01	9.93E-01	2.21E-08	7.34E-06	1.51E-04	1.56E+00	2.01E+00	6.05E+00	2.26E-07	7.79E-05	9.04E-04
1960	1.61E-01	2.03E-01	1.10E+00	2.50E-08	8.31E-06	1.71E-04	1.73E+00	2.23E+00	6.72E+00	2.56E-07	8.82E-05	1.02E-03
1961	1.78E-01	2.24E-01	1.22E+00	2.65E-08	8.83E-06	1.82E-04	1.91E+00	2.46E+00	7.42E+00	2.72E-07	9.37E-05	1.09E-03
1962	1.66E-01	2.09E-01	1.14E+00	2.44E-08	8.13E-06	1.68E-04	1.79E+00	2.30E+00	6.94E+00	2.50E-07	8.63E-05	1.00E-03
1963	1.81E-01	2.28E-01	1.24E+00	2.59E-08	8.62E-06	1.77E-04	1.95E+00	2.51E+00	7.55E+00	2.65E-07	9.14E-05	1.06E-03
1964	2.42E-01	3.05E-01	1.66E+00	3.42E-08	1.14E-05	2.35E-04	2.61E+00	3.36E+00	1.01E+01	3.51E-07	1.21E-04	1.40E-03
1965	1.84E-01	2.32E-01	1.26E+00	2.44E-08	8.11E-06	1.67E-04	1.98E+00	2.55E+00	7.68E+00	2.50E-07	8.60E-05	9.98E-04
1966	1.70E-01	2.14E-01	1.17E+00	2.25E-08	7.49E-06	1.54E-04	1.83E+00	2.36E+00	7.11E+00	2.31E-07	7.95E-05	9.22E-04
1967	2.37E-01	2.99E-01	1.63E+00	3.15E-08	1.05E-05	2.16E-04	2.56E+00	3.29E+00	9.92E+00	3.23E-07	1.11E-04	1.29E-03
1968	2.37E-01	2.99E-01	1.63E+00	3.15E-08	1.05E-05	2.16E-04	2.56E+00	3.29E+00	9.92E+00	3.23E-07	1.11E-04	1.29E-03
1969	2.37E-01	2.99E-01	1.63E+00	3.15E-08	1.05E-05	2.16E-04	2.56E+00	3.29E+00	9.92E+00	3.23E-07	1.11E-04	1.29E-03
1970	2.37E-01	2.99E-01	1.63E+00	3.15E-08	1.05E-05	2.16E-04	2.56E+00	3.29E+00	9.92E+00	3.23E-07	1.11E-04	1.29E-03
1971	2.37E-01	2.99E-01	1.63E+00	3.15E-07	4.03E-07	2.29E-06	2.56E+00	3.29E+00	9.92E+00	3.23E-06	4.27E-06	1.37E-05
1972	2.37E-01	2.99E-01	1.63E+00	3.15E-07	4.03E-07	2.29E-06	2.56E+00	3.29E+00	9.92E+00	3.23E-06	4.27E-06	1.37E-05
1973	2.37E-01	2.99E-01	1.63E+00	3.15E-07	4.03E-07	2.29E-06	2.56E+00	3.29E+00	9.92E+00	3.23E-06	4.27E-06	1.37E-05
1974	2.37E-01	2.99E-01	1.63E+00	6.29E-08	8.06E-08	4.59E-07	2.56E+00	3.29E+00	9.92E+00	6.45E-07	8.55E-07	2.74E-06
1975	2.37E-01	2.99E-01	1.63E+00	6.29E-08	8.06E-08	4.59E-07	2.56E+00	3.29E+00	9.92E+00	6.45E-07	8.55E-07	2.74E-06
1976	2.37E-01	2.99E-01	1.63E+00	6.29E-08	8.06E-08	4.59E-07	2.56E+00	3.29E+00	9.92E+00	6.45E-07	8.55E-07	2.74E-06
1977	2.37E-01	2.99E-01	1.63E+00	6.29E-08	8.06E-08	4.59E-07	2.56E+00	3.29E+00	9.92E+00	6.45E-07	8.55E-07	2.74E-06
1978	2.37E-01	2.99E-01	1.63E+00	6.29E-08	8.06E-08	4.59E-07	2.56E+00	3.29E+00	9.92E+00	6.45E-07	8.55E-07	2.74E-06
1979	2.37E-01	2.99E-01	1.63E+00	6.29E-08	8.06E-08	4.59E-07	2.56E+00	3.29E+00	9.92E+00	6.45E-07	8.55E-07	2.74E-06

[a] *Blank cells = data not available and/or no spraying occurred.*

[b] *PHED = Pesticide Handler Exposure Database.*

[c] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[d] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 4.38. *Estimates of Ministry of Natural Resources occupational exposure (aerial) to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Year	PHED[b] data—applicator—aerial, fixed wing (µg/kg/day)						PHED data—flagman—aerial, fixed wing (µg/kg/day)						PHED data—mixer/loader—aerial, fixed wing (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[c]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1953	6.62E-04	6.62E-04	6.62E-04	3.31E-11	8.61E-09	3.11E-08	1.76E-03	1.91E-03	1.81E-03	8.81E-11	2.49E-08	8.49E-08	3.54E-03	4.47E-03	2.43E-02	1.77E-10	5.81E-08	1.14E-06
1954																		
1955																		
1956	1.93E-02	1.93E-02	1.93E-02	9.64E-10	2.51E-07	9.06E-07	5.13E-02	5.58E-02	5.26E-02	2.57E-09	7.25E-07	2.47E-06	1.03E-01	1.30E-01	7.08E-01	5.16E-09	1.69E-06	3.33E-05
1957	1.13E-01	1.13E-01	1.13E-01	5.66E-09	1.47E-06	5.32E-06	3.01E-01	3.28E-01	3.09E-01	1.51E-08	4.26E-06	1.45E-05	6.06E-01	7.64E-01	4.16E+00	3.03E-08	9.94E-06	1.95E-04
1958	4.77E-02	4.77E-02	4.77E-02	2.38E-09	6.20E-07	2.24E-06	1.27E-01	1.38E-01	1.30E-01	6.35E-09	1.79E-06	6.11E-06	2.55E-01	3.22E-01	1.75E+00	1.28E-08	4.18E-06	8.22E-05
1959	7.48E-02	7.48E-02	7.48E-02	3.74E-09	9.72E-07	3.51E-06	1.99E-01	2.16E-01	2.04E-01	9.95E-09	2.81E-06	9.59E-06	4.00E-01	5.05E-01	2.74E+00	2.00E-08	6.56E-06	1.29E-04
1960	4.10E-02	4.10E-02	4.10E-02	2.05E-09	5.33E-07	1.93E-06	1.09E-01	1.19E-01	1.12E-01	5.45E-09	1.54E-06	5.25E-06	2.19E-01	2.77E-01	1.50E+00	1.10E-08	3.60E-06	7.07E-05
1961	8.52E-02	8.52E-02	8.52E-02	4.26E-09	1.11E-06	4.00E-06	2.27E-01	2.46E-01	2.32E-01	1.13E-08	3.20E-06	1.09E-05	4.56E-01	5.75E-01	3.13E+00	2.28E-08	7.47E-06	1.47E-04
1962	3.61E-02	3.61E-02	3.61E-02	1.81E-09	4.70E-07	1.70E-06	9.62E-02	1.04E-01	9.85E-02	4.81E-09	1.36E-06	4.63E-06	1.93E-01	2.44E-01	1.33E+00	9.67E-09	3.17E-06	6.23E-05
1963	9.42E-02	9.42E-02	9.42E-02	4.71E-09	1.22E-06	4.43E-06	2.51E-01	2.72E-01	2.57E-01	1.25E-08	3.54E-06	1.21E-05	5.04E-01	6.36E-01	3.46E+00	2.52E-08	8.27E-06	1.62E-04
1964	9.54E-02	9.54E-02	9.54E-02	4.77E-09	1.24E-06	4.49E-06	2.54E-01	2.76E-01	2.60E-01	1.27E-08	3.59E-06	1.22E-05	5.11E-01	6.44E-01	3.50E+00	2.55E-08	8.37E-06	1.65E-04
1965	3.95E-02	3.95E-02	3.95E-02	1.97E-09	5.13E-07	1.86E-06	1.05E-01	1.14E-01	1.08E-01	5.26E-09	1.49E-06	5.06E-06	2.11E-01	2.67E-01	1.45E+00	1.06E-08	3.47E-06	6.81E-05
1966	1.02E-02	1.02E-02	1.02E-02	5.08E-10	1.32E-07	4.77E-07	2.70E-02	2.94E-02	2.77E-02	1.35E-09	3.82E-07	1.30E-06	5.43E-02	6.85E-02	3.72E-01	2.72E-09	8.91E-07	1.75E-05
1967	2.08E-02	2.08E-02	2.08E-02	1.04E-09	2.70E-07	9.77E-07	5.53E-02	6.01E-02	5.67E-02	2.77E-09	7.81E-07	2.67E-06	1.11E-01	1.40E-01	7.63E-01	5.56E-09	1.82E-06	3.58E-05
1968																		
1969	1.37E-02	1.37E-02	1.37E-02	6.84E-10	1.78E-07	6.43E-07	3.64E-02	3.96E-02	3.73E-02	1.82E-09	5.14E-07	1.75E-06	7.33E-02	9.23E-02	5.02E-01	3.66E-09	1.20E-06	2.36E-05
1970																		
1971																		
1972																		
1973	6.53E-03	6.53E-03	6.53E-03	3.27E-09	3.27E-09	3.27E-09	1.74E-02	1.89E-02	1.78E-02	8.69E-09	9.44E-09	8.91E-09	3.50E-02	4.41E-02	2.40E-01	1.75E-08	2.20E-08	1.20E-07
1974																		
1975																		
1976																		
1977																		
1978																		
1979	2.65E-03	2.65E-03	2.65E-03	2.65E-10	2.65E-10	2.65E-10	7.05E-03	7.66E-03	7.22E-03	7.05E-10	7.66E-10	7.22E-10	1.42E-02	1.79E-02	9.72E-02	1.42E-09	1.79E-09	9.72E-09

[a] *Blank cells = data not available and/or no spraying occurred.*
[b] *PHED = Pesticide Handler Exposure Database.*
[c] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[d] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 4.39. *Estimates of Ministry of Natural Resources occupational exposure (ground-vehicle) to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Year	PHED[b] data—mixer/loader (ground only) total (µg/kg/day)						PHED data—right-of-way applicator total (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[c]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1959	1.35E-03	1.70E-03	9.24E-03	6.74E-11	2.21E-08	4.34E-07	1.45E-02	1.87E-02	5.64E-02	7.27E-10	2.43E-07	2.65E-06[d]
1960												
1961												
1962												
1963												
1964												
1965												
1966												
1967												
1968												
1969												
1970												
1971	2.46E-04	3.10E-04	1.69E-03	2.46E-11	3.10E-11	1.69E-10	2.65E-03	3.42E-03	1.03E-02	2.65E-10	3.42E-10	1.03E-09
1972												
1973												
1974												
1975												
1976												
1977												
1978												
1979												

[a] *Blank cells = data not available and/or no spraying occurred.*
[b] *PHED = Pesticide Handler Exposure Database.*
[c] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[d] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 4.40. *Estimates of Ministry of Natural Resources occupational exposure (ground-backpack) to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Year	PHED[b] data—mixer/loader (ground only) total (µg/kg/day)						PHED data—right-of-way applicator total (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[c]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1948	3.77E-03	7.52E-03	7.52E-03	1.88E-10	9.77E-08	3.53E-07	4.46E-03	8.90E-03	8.90E-03	2.23E-10	1.16E-07	4.18E-07
1949												
1950												
1951												
1952												
1953	5.30E-01	1.06E+00	1.06E+00	2.65E-08	1.37E-05[d]	4.97E-05	6.28E-01	1.25E+00	1.25E+00	3.14E-08	1.63E-05	5.89E-05
1954												
1955												
1956												
1957	6.88E-01	1.37E+00	1.37E+00	3.44E-08	1.79E-05	6.45E-05	8.15E-01	1.63E+00	1.63E+00	4.08E-08	2.11E-05	7.64E-05
1958	7.54E-01	1.50E+00	1.50E+00	3.77E-08	1.95E-05	7.07E-05	8.93E-01	1.78E+00	1.78E+00	4.46E-08	2.31E-05	8.37E-05
1959	1.01E+00	2.01E+00	2.01E+00	5.03E-08	2.61E-05	9.43E-05	1.19E+00	2.38E+00	2.38E+00	5.96E-08	3.09E-05	1.12E-04
1960	9.90E-01	1.97E+00	1.97E+00	4.95E-08	2.57E-05	9.28E-05	1.17E+00	2.34E+00	2.34E+00	5.86E-08	3.04E-05	1.10E-04
1961	2.46E-01	4.91E-01	4.91E-01	1.23E-08	6.39E-06	2.31E-05	2.92E-01	5.82E-01	5.82E-01	1.46E-08	7.56E-06	2.73E-05
1962	2.10E+00	4.19E+00	4.19E+00	1.05E-07	5.44E-05	1.97E-04	2.48E+00	4.96E+00	4.96E+00	1.24E-07	6.44E-05	2.33E-04
1963	1.07E-01	2.13E-01	2.13E-01	5.34E-09	2.77E-06	1.00E-05	1.26E-01	2.52E-01	2.52E-01	6.32E-09	3.28E-06	1.19E-05
1964	1.70E+00	3.39E+00	3.39E+00	8.49E-08	4.40E-05	1.59E-04	2.01E+00	4.01E+00	4.01E+00	1.00E-07	5.21E-05	1.88E-04
1965	7.32E-01	1.46E+00	1.46E+00	3.66E-08	1.90E-05	6.87E-05	8.67E-01	1.73E+00	1.73E+00	4.34E-08	2.25E-05	8.13E-05
1966	2.26E-01	4.51E-01	4.51E-01	1.13E-08	5.86E-06	2.12E-05	2.68E-01	5.34E-01	5.34E-01	1.34E-08	6.94E-06	2.51E-05
1967	1.05E+00	2.09E+00	2.09E+00	5.23E-07	1.04E-06	1.04E-06	1.24E+00	2.47E+00	2.47E+00	6.20E-07	1.24E-06	1.24E-06
1968	4.52E-01	9.02E-01	9.02E-01	2.26E-07	4.51E-07	4.51E-07	5.36E-01	1.07E+00	1.07E+00	2.68E-07	5.34E-07	5.34E-07
1969												
1970												
1971												
1972												
1973	5.53E-02	1.10E-01	1.10E-01	5.53E-09	1.10E-08	1.10E-08	6.55E-02	1.31E-01	1.31E-01	6.55E-09	1.31E-08	1.31E-08
1974	1.13E-01	2.26E-01	2.26E-01	1.13E-08	2.26E-08	2.26E-08	1.34E-01	2.67E-01	2.67E-01	1.34E-08	2.67E-08	2.67E-08

[a] *Blank cells = data not available and/or no spraying occurred.*
[b] *PHED = Pesticide Handler Exposure Database.*
[c] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[d] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

4.8.3. Non-occupational exposure estimates

4.8.3.1. Estimating recreational visitor exposure

Areas of Ontario that have been affected by herbicide spraying are often used by members of the public for recreating. The panel assumed an individual would spend four hours/day, 14 days/year involved in recreational activities in areas where aerial/remote spray operations have occurred (MNR and Ontario Hydro). During this time, individuals were assumed to come into contact with chemicals of concern via four main exposure pathways: incidental soil ingestion, direct dermal contact with surface soils, inhalation of re-suspended soil, and consumption of wild berries. Using the receptor characteristics and activity patterns described in tables 4.27 and 4.30, respectively, and the soil and wild berry concentrations presented in tables 4.16 and 4.21, the panel estimated receptor exposure to 2,4,5-T and TCDD while spending time in an remote spray area as shown below.

The panel calculated exposure via incidental ingestion of soil as follows:

$$Dose_{oral} = \frac{C_{soil} \times SIR \times AF_{soil} \times EF}{AT \times BW} \qquad \text{(Equation 4.18)}$$

Where:

$Dose_{oral}$	=	absorbed dose via incidental soil ingestion (µg/kg BW/day)
C_{soil}	=	soil concentration at 1 cm depth (µg/g)
AF_{soil}	=	soil bioaccessibility factor (unitless)
SIR	=	incidental soil ingestion rate (g/d)
EF	=	exposure frequency (days/year)
AT	=	averaging time (365 days)
BW	=	body weight (kg)

The recreational visitor was also assumed to be exposed to the chemicals of concern through direct dermal contact with affected soil. The panel calculated exposure to 2,4,5-T and TCDD via dermal contact with soil as follows:

$$Dose_{dermal} = \frac{C_{soil} \times (SA_{whole\ body} \times ADF_{whole\ body}) \times AF_{dermal} \times EF}{AT \times BW} \qquad \text{(Equation 4.19)}$$

Where:

$Dose_{dermal}$	=	absorbed dose from direct dermal contact with soil (µg/kg BW/day)
C_{soil}	=	soil concentration at 1 cm depth (µg/g)
$SA_{whole\ body}$	=	surface area of exposed whole body (cm^2)
$ADF_{whole\ body}$	=	soil adherence factor—whole body (g/cm^2—day)
AF_{dermal}	=	dermal absorption factor (unitless)
EF	=	exposure frequency (d/yr)
AT	=	averaging time (365 days)
BW	=	body weight (kg)

The recreational visitor was also assumed to be exposed to chemicals of concern through the inhalation of affected soils. The panel calculated exposure to 2,4,5-T and TCDD via inhalation of soil as follows:

$$Dose_{inhalation} = \frac{C_{soil} \times IR \times P_{air} \times AF_{inhalation} \times EF}{AT \times BW} \qquad \text{(Equation 4.20)}$$

Where:

$Dose_{inhalation}$ = absorbed dose via inhalation of soil/dust (µg/kg BW/day)
C_{soil} = soil concentration at 1 cm depth (µg/g)
P_{air} = particulate concentration in air (7.6×10^{-7} g/m^3)
$AF_{inhalation}$ = relative absorption factor for inhalation (unitless)
IR = inhalation rate (m^3/day)
EF = exposure frequency (24 hours/day x 275 days/year)
AT = averaging time (365 days)
BW = body weight (kg)

The recreational visitor was also assumed to be exposed to chemicals of concern via consumption of local wild berries. The following equation was used to predict exposures of the recreational visitor to the chemicals as a result of consuming wild berries:

$$Dose_{berries} = \frac{C_{berries} \times WBC \times AF_{gut}}{BW} \qquad \text{(Equation 4.21)}$$

Where:

$Dose_{berries}$ = absorbed dose via consumption of wild berries (µg/kg BW/day)
$C_{berries}$ = concentration in wild berries (µg/kg)
WBC = wild berry consumption rate (g/day)
AF_{gut} = relative absorption factor in the gut (unitless)
BW = body weight (kg)

The panel then calculated the total exposure of a recreational visitor as the sum of incidental soil/dust ingestion, direct dermal contact with surface soils, inhalation of re-suspended soil, and consumption of wild berries exposure estimates. The panel also considered recreational receptor consumption of fish and wild game, as calculated in sections 4.4.4.5 and 4.4.4.6.

Table 4.41. *Estimate of recreational visitor exposures to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

	Unit	Ontario Hydro				MNR			
		2,4,5-T	TCDD			2,4,5-T	TCDD		
			Low[a]	Central	High		Low	Central	High
Receptor parameter									
Body weight	kg	70.7	70.7	70.7	70.7	70.7	70.7	70.7	70.7
Amount of soil consumed	mg/day	50	50	50	50	50	50	50	50
Exposed surface area	cm^2	4343	4343	4343	4343	4343	4343	4343	4343
Soil adherence factor—body	g/cm^2 / event	0.00007	0.00007	0.00007	0.00007	0.00007	0.00007	0.00007	0.00007
Inhalation rate	m^3/day	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6
particulate concentration in air	g/m^3	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07
Hours per day	hr	4	4	4	4	4	4	4	4
Days per year (direct soil)	d/yr	14	14	14	14	14	14	14	14
Averaging time	d/yr	365	365	365	365	365	365	365	365
Wild berry consumption rate	g/day	5.2	5.2	5.2	5.2	5.2	5.2	5.2	5.2
Local fish consumption rate	g/day	24.42	24.42	24.42	24.42	24.42	24.42	24.42	24.42
Wild game consumption rate	g/day	1.8	1.8	1.8	1.8	1.7	1.7	1.7	1.7
Chemical parameter									
Soil concentration—ingestion/ inhalation/dermal	µg/g	9.41E-01	4.72E-07	1.49E-05	6.43E-05	4.03E-01	2.02E-08	5.25E-06	1.90E-05
Wild berry concentration	µg/g	4.36E-03	2.07E-11	6.51E-10	2.82E-09	1.87E-03	8.84E-13	2.30E-10	8.31E-10
Fish concentration	µg/g	NA[b]	9.66E-10	2.51E-07	9.08E-07	NA	4.14E-10	1.08E-07	3.89E-07
Wild game concentration	µg/g	NA	4.03E-10	1.5E-08	6.3E-08	NA	2.11E-11	5.48E-09	1.98E-08
Soil bioavailability	unitless	1	0.34	0.34	0.34	1	0.34	0.34	0.34
Dermal RAF	unitless	0.03	0.08	0.08	0.08	0.03	0.08	0.08	0.08
Exposure estimate									
Soil ingestion	µg/kg/day	2.55E-05	4.36E-12	1.37E-10	5.93E-10	1.09E-05	1.86E-13	4.84E-11	1.75E-10
Soil inhalation	µg/kg/day	6.44E-09	3.23E-15	1.02E-13	4.40E-13	2.76E-09	1.38E-16	3.59E-14	1.30E-13
Soil dermal	µg/kg/day	3.95E-06	5.86E-12	1.84E-10	7.98E-10	1.69E-06	2.50E-13	6.51E-11	2.35E-10
Wild berry	µg/kg/day	3.21E-04	1.52E-12	4.79E-11	2.07E-10	1.37E-04	6.50E-14	1.69E-11	6.11E-11
Fish and wild game	µg/kg/day	NA	3.85E-11	8.87E-09	3.22E-08	NA	1.46E-11	3.79E-09	1.37E-08
Exposure									
Recreational visitor (adult)	µg/kg/day	3.50E-04	1.17E-11	3.70E-10	1.60E-09	1.50E-04	5.02E-13	1.30E-10	4.71E-10
Hunter/angler (total)	µg/kg/day	NA	5.02E-11	9.24E-09	3.38E-08	NA	1.51E-11	3.92E-09	1.42E-08

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[b] *NA = not applicable.*

4.8.3.2. Estimating resident exposure

Parts of Ontario affected by herbicide applications are close to residential areas. Thus, both adults and children of various ages could have been exposed to the chemicals of concern during their time at home. Toddlers tend to be the most sensitive receptor group due to a higher exposure per body weight than other age groups. It was assumed that a toddler living in a residence would spend 24 hours/day, 365 days/year at this location. An average three months of snow cover was assumed for Ontario (MOE 2011). During this time, toddlers were assumed to come into contact with chemicals of concern via these main exposure pathways: incidental soil/dust ingestion, direct dermal contact with surface soils, inhalation of re-suspended soil, and consumption of wild berries. Toddlers with hunter/angler parents were also assumed to consumed fish and wild game from spray areas. Using the receptor characteristics and activity patterns described in tables 4.27 and 4.30, respectively, and maximum soil, fish, wild game, and wild berry concentrations presented in tables 4.16 to 4.25, the panel estimated toddler exposure to 2,4,5-T and TCDD while spending time in proximity to a spray area as shown below.

The panel calculated toddler exposure via incidental ingestion of soil in proximity to a spray area as follows:

$$Dose_{oral} = \frac{C_{soil} \times SIR \times AF_{soil} \times EF}{AT \times BW} \quad \text{(Equation 4.22)}$$

Where:

$Dose_{oral}$	=	absorbed dose via incidental soil ingestion (µg/kg BW/day)
C_{soil}	=	soil concentration at 1 cm depth (µg/g)
AF_{soil}	=	oil bioaccessibility factor (unitless)
SIR	=	incidental soil ingestion rate (g/d)
EF	=	exposure frequency (days/year)
AT	=	averaging time (365 days)
BW	=	body weight (kg)

The resident toddler was also assumed to be exposed to chemicals of concern through direct dermal contact with affected soil. The panel calculated exposure from dermal contact with soil in proximity to a spray area as follows:

$$Dose_{dermal} = \frac{C_{soil} \times (SA_{whole\ body} \times ADF_{whole\ body}) \times AF_{dermal} \times EF}{AT \times BW} \quad \text{(Equation 4.23)}$$

Where:

$Dose_{dermal}$	=	absorbed dose from direct dermal contact with soil (µg/kg BW/day)
C_{soil}	=	soil concentration at 1 cm depth (µg/g)
$SA_{whole\ body}$	=	surface area of exposed whole body (cm^2)
$ADF_{whole\ body}$	=	soil adherence factor—whole body (g/cm^2—day)
AF_{dermal}	=	dermal absorption factor (unitless)
EF	=	exposure frequency (d/yr)
AT	=	averaging time (365 days)
BW	=	body weight (kg)

The resident toddler was also assumed to be exposed to chemicals of concern via inhalation of affected soils. The panel calculated exposure via inhalation of soil in proximity to a spray area as follows:

$$Dose_{inhalation} = \frac{C_{soil} \times IR \times P_{air} \times AF_{inhalation} \times EF}{AT \times BW} \quad \text{(Equation 4.24)}$$

Where:

$Dose_{inhalation}$ = absorbed dose via inhalation of soil/dust (µg/kg BW/day)
C_{soil} = soil concentration at 1 cm depth (µg/g)
P_{air} = particulate concentration in air (7.6×10^{-7} g/m^3)
$AF_{inhalation}$ = relative absorption factor for inhalation (unitless)
IR = inhalation rate (m^3/day)
EF = exposure frequency (24 hours/day x 275 days/year)
AT = averaging time (365 days)
BW = body weight (kg)

The resident toddler was also assumed to be exposed to chemicals of concern via consumption of local wild berries. The panel used the following equation to predict resident toddler exposure to chemicals of concern via consuming wild berries:

$$Dose_{berries} = \frac{C_{berries} \times WBC \times AF_{gut}}{BW} \quad \text{(Equation 4.25)}$$

Where:

$Dose_{berries}$ = absorbed dose via consumption of wild berries (µg/kg BW/day)
$C_{berries}$ = concentration in wild berries (µg/kg)
WBC = wild berry consumption rate (g/day)
AF_{gut} = relative absorption factor in the gut (unitless)
BW = body weight (kg)

Table 4.42 presented the total exposure of a resident as a result of incidental soil/dust ingestion, direct dermal contact with surface soils, inhalation of re-suspended soil, and consumption of wild berries in proximity to a spray area. The panel also considered residential toddler consumption of fish and wild game, as calculated in sections 4.4.4.5 and 4.4.4.6.

Table 4.42. *Estimates of residential exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

	Unit	Ontario Hydro				MTO			
		2,4,5-T	TCDD			2,4,5-T	TCDD		
			Low[a]	Central	High		Low	Central	High
Receptor parameter									
Body weight (toddler)	kg	16.5	16.5	16.5	16.5	16.5	16.5	16.5	16.5
Amount of soil consumed	mg/day	200	200	200	200	200	200	200	200
Exposed surface area	cm^2	1745	1745	1745	1745	1745	1745	1745	1745
Soil adherence factor—body	g/cm^2 / event	0.0002	0.0002	0.0002	0.0002	0.0002	0.0002	0.0002	0.0002
Inhalation rate	m^3/day	8.3	8.3	8.3	8.3	8.3	8.3	8.3	8.3
Particulate concentration in air	g/m^3	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07
Hours per day	hr	24	24	24	24	24	24	24	24
Days per year (direct soil)	d/yr	275	275	275	275	275	275	275	275
Averaging time	d/yr	365	365	365	365	365	365	365	365
Wild berry consumption rate	g/day	1.2	1.2	1.2	1.2	1.2	1.2	1.2	1.2
Local fish consumption rate	g/day	21.3	21.3	21.3	21.3	21.3	21.3	21.3	21.3
Wild game consumption rate	g/day	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8
Chemical parameter									
Soil concentration—ingestion/inhalation/ dermal	µg/g	5.23E-01	2.62E-07	8.26E-06	3.58E-05	4.51E-01	4.00E-07	2.00E-05	1.01E-04
Wild berry concentration	µg/g	2.42E-03	1.15E-11	3.62E-10	1.57E-09	2.09E-03	1.75E-11	8.74E-10	4.41E-09
Fish concentration	µg/g	NA[b]	9.66E-10	2.51E-07	9.08E-07	NA	2.49E-09	6.47E-07	2.34E-06
Wild game concentration	µg/g	NA	4.03E-10	1.50E-08	6.30E-08	NA			
Soil bioavailability	unitless	1	0.34	0.34	0.34	1	0.34	0.34	0.34
Dermal RAF	unitless	0.14	0.37	0.37	0.37	0.14	0.37	0.37	0.37
Exposure estimate									
Soil ingestion	µg/kg/day	4.78E-03	8.15E-10	2.56E-08	1.11E-07	4.12E-03	1.24E-09	6.20E-08	3.13E-07
Soil inhalation	µg/kg/day	1.51E-07	7.56E-14	2.38E-12	1.03E-11	1.30E-07	1.15E-13	5.75E-12	2.90E-11
Soil dermal	µg/kg/day	1.19E-03	1.57E-09	4.93E-08	2.14E-07	1.03E-03	2.39E-09	1.19E-07	6.01E-07
Wild berry	µg/kg/day	1.76E-04	8.36E-13	2.63E-11	1.14E-10	1.52E-04	1.27E-12	6.36E-11	3.21E-10
Fish and wild game	µg/kg/day	NA	1.35E-10	3.28E-08	1.19E-07	NA	3.21E-10	8.35E-08	3.02E-07
Exposure									
Resident (toddler)	µg/kg/day	6.15E-03	2.38E-09	7.50E-08	3.25E-07	5.30E-03	3.63E-09	1.81E-07	9.14E-07
Hunter/angler child (toddler)	µg/kg/day	NA	2.52E-09	1.08E-07	4.43E-07	NA	3.95E-09	2.65E-07	1.22E-06

[a] Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.

[b] NA = not applicable.

4.8.3.3. Estimating angler exposure

Portions of Ontario affected by herbicide applications are open to the public for sport fishing. In addition, traditional First Nations fishing lands may have been sprayed with herbicides. As a result, anglers could have been exposed to the chemicals of concern via consumption of their catch. While fishing, anglers likely would have come into contact with the chemicals of concern via other exposure pathways, including incidental soil/dust ingestion, direct dermal contact with surface soils, and inhalation of re-suspended soil. Risks from these pathways were assessed for the recreational visitor and resident receptors so were not repeated here. Using the receptor characteristics described in Table 4.27 and fish concentrations presented in Table 4.23, the panel estimated angler and First Nations exposure to TCDD via consuming fish as shown below.

$$EXP_{fish} = \frac{C_{fish} \times FC \times F \times AF_{oral}}{BW} \qquad \text{(Equation 4.26)}$$

Where:

EXP_{fish}	=	absorbed dose via consumption of local fish (µg/kg bw/day)
C_{fish}	=	concentration of TCDD in local fish (µg/g)
FC	=	local fish consumption rate (g/day)
F	=	fraction of fish from spray area (unitless)
AF_{oral}	=	bioavailability of TCDD in the gut (unitless)
BW	=	body weight (kg)

The total exposure of anglers and First Nations receptors to TCDD via consumpion of local fish is presented in Table 4.43.

Table 4.43. *Estimates of fish consumption exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (µg/kg/day).*

	Ontario Hydro			Ministry of Natural Resources		
	Low[a]	Central	High	Low	Central	High
Angler-fish consumer	3.3E-11	8.7E-09	3.1E-08	1.4E-11	3.7E-09	1.3E-08
First Nations-fish consumer	7.5E-10	2.0E-07	7.1E-07	3.2E-10	8.4E-08	3.0E-07

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

4.8.3.4. Estimating hunter exposure

Parts of Ontario affected by herbicide applications are open to the public for hunting. In addition, traditional First Nations hunting grounds may have been sprayed. Thus, hunters could have been exposed to the chemicals of concern through consumption of their catch. Hunters likely would have come into contact with chemicals of concern via other exposure pathways, including incidental soil/dust ingestion, direct dermal contact with surface soils, and inhalation of re-suspended soil. Risks from these pathways were assessed for the recreational visitor and resident receptors so were not repeated here. Daily intake rates of wild game consistent with those used in Health Canada (2010) were used. Using the receptor characteristics described in Table 4.27 and wild game concentrations presented in Table 4.25, the panel estimated hunter and First Nations receptor exposure to TCDD via consuming wild game as follows:

$$EXP_{game} = \frac{C_{game} \times WGC \times F \times AF_{oral}}{BW} \quad \text{(Equation 4.27)}$$

Where:

EXP_{game}	=	absorbed dose via consumption of local wild game (µg/kg bw/day)
C_{game}	=	concentration of TCDD in local wild game (µg/g)
WGC	=	local wild game consumption rate (g/day)
F	=	fraction of game from spray area (unitless)
AF_{oral}	=	bioavailability of TCDD in the gut (unitless)
BW	=	body weight (kg)

The total exposure of hunter and First Nations receptors to TCDD as a result of consumption of local wild game are provided in Table 4.44.

Table 4.44. *Estimates of wild game consumption exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (µg/kg/day).*

Ontario Hydro	Moose			White-tailed deer		
	Low[a]	Central	High	Low	Central	High
Hunter-game consumer	5.1E-11	1.9E-09	8.0E-09	2.2E-11	1.3E-09	5.0E-09
First Nations-game consumer	7.7E-10	2.9E-08	1.2E-07	3.4E-10	1.9E-08	7.5E-08
Ministry of Natural Resources	**Moose**			**White-tailed deer**		
	Low	Central	High	Low	Central	High
Hunter-game consumer	2.7E-12	7.0E-10	2.5E-09	1.9E-12	4.9E-10	1.8E-09
First Nations-game consumer	4.0E-11	1.0E-08	3.8E-08	2.8E-11	7.4E-09	2.7E-08

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

4.8.3.5. Estimating bystander exposure to spray drift

Individuals (e.g., adults and children) may have been in the vicinity of herbicide spray activities in Ontario. A preschool child was assumed to be standing in close proximity to an intended spray area and to come into direct contact with aerial spray drift (MNR) or overspray (MTO/ Ontario Hydro). Exposures via inhalation and direct dermal contact were evaluated at different distances (scenario dependent) from the spray area. These exposures were considered to occur over a very short time. Individuals were assumed to be standing in the worst possible location at the worst possible time relative to coming into direct contact with herbicide overspray.

Two situations were considered: aerial (MNR/Ontario Hydro) and ground spray (MTO/Ontario Hydro).

It was assumed that a bystander may be located 30 m from an area where ground-based spraying was occurring and 500 m from an area where aerial spraying was underway. Tables 4.45 to 4.46 present bystander exposure estimates.

Direct inhalation

Acute inhalation risks for bystanders were evaluated at different distances (scenario dependent). Application rates were assumed to represent instantaneous air concentrations as the herbicide passes through the breathing zone (assumed to be 1 m^3 for a toddler). The panel used generic AgDrift drift curves to estimate the percentage of herbicide at off-target locations and estimated exposure as follows:

$$Dose_{inhalation} = \frac{AR \times DL \times IR \times CF \times AT \times AF_{inhalation}}{BW} \quad \text{(Equation 4.28)}$$

Where:

$Dose_{inhalation}$	=	absorbed dose via inhalation of overspray (µg/kg/d)
AR	=	contaminant application rate (g/m^2)
DL	=	drift loss fraction (unitless)
IR	=	inhalation rate (m^3/day)
CF	=	conversion factors (1,000,000 µg/g and 1 m^2/m^3)
$AF_{inhalation}$	=	inhalation absorption factor (unitless)
AT	=	averaging time—assumes 1 minute of exposure (per day)
BW	=	body weight (kg)

Direct dermal contact with overspray

Exposures via direct dermal contact with overspray were also evaluated at different distances (scenario specific) from the spray areas. As a worst-case acute exposure scenario, a bystander (a preschool child) was assumed to be standing close to an intended spray area while coming into direct dermal contact with aerial spray drift or overspray. This exposure scenario is considered short term (or acute) with herbicide remaining on the skin no more than one hour.

The panel calculated an absorbed dose resulting from direct dermal contact with overspray as follows:

$$Dose_{dermal} = \frac{AR \times DL \times SA \times CF \times AF_{dermal}}{BW} \quad \text{(Equation 4.29)}$$

Where:

$Dose_{dermal}$	=	absorbed dose via direct contact with overspray (µg/kg/d)
AR	=	contaminant application rate (g/m^2)
DL	=	drift loss fraction (unitless)
SA	=	exposed skin surface area (m^2)
CF	=	conversion factor (1,000,000 µg/g)
AF_{dermal}	=	dermal absorption factor (unitless)
BW	=	body weight (kg)

Table 4.45. *Estimates of bystander exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)—1 day per year direct spray (Ontario Hydro).*

Year	Ground-based exposure at 30 m (µg/kg/day)				Aerial (fixed)-based exposure at 500 m (µg/kg/day)				Aerial (rotary)-based exposure at 500 m (µg/kg/day)			
	2,4,5-T	TCDD			2,4,5-T	TCDD			2,4,5-T	TCDD		
		Low[a]	Central	High		Low	Central	High		Low	Central	High
1948	4.91E+00	3.26E-07	8.48E-05	3.06E-04[b]	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1949	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1950	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1951	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1952	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1953	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1954	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1955	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1956	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1957	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1958	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1959	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1960	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1961	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1962	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1963	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1964	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1965	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1966	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1967	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1968	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1969	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1970	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1971	4.91E+00	3.26E-06	3.26E-06	3.26E-06	1.23E+00	2.57E-07	6.17E-07	9.25E-07	2.45E+00	1.63E-06	1.63E-06	1.63E-06
1972	4.91E+00	3.26E-06	3.26E-06	3.26E-06	1.23E+00	2.57E-07	6.17E-07	9.25E-07	2.45E+00	1.63E-06	1.63E-06	1.63E-06
1973	4.91E+00	3.26E-06	3.26E-06	3.26E-06	1.23E+00	2.57E-07	6.17E-07	9.25E-07	2.45E+00	1.63E-06	1.63E-06	1.63E-06
1974	4.91E+00	6.52E-07	6.52E-07	6.52E-07	1.23E+00	5.14E-08	1.23E-07	1.85E-07	2.45E+00	3.26E-07	3.26E-07	3.26E-07
1975	4.91E+00	6.52E-07	6.52E-07	6.52E-07	1.23E+00	5.14E-08	1.23E-07	1.85E-07	2.45E+00	3.26E-07	3.26E-07	3.26E-07
1976	4.91E+00	6.52E-07	6.52E-07	6.52E-07	1.23E+00	5.14E-08	1.23E-07	1.85E-07	2.45E+00	3.26E-07	3.26E-07	3.26E-07
1977	4.91E+00	6.52E-07	6.52E-07	6.52E-07	1.23E+00	5.14E-08	1.23E-07	1.85E-07	2.45E+00	3.26E-07	3.26E-07	3.26E-07
1978	4.91E+00	6.52E-07	6.52E-07	6.52E-07	1.23E+00	5.14E-08	1.23E-07	1.85E-07	2.45E+00	3.26E-07	3.26E-07	3.26E-07
1979	4.91E+00	6.52E-07	6.52E-07	6.52E-07	1.23E+00	5.14E-08	1.23E-07	1.85E-07	2.45E+00	3.26E-07	3.26E-07	3.26E-07

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[b] *Coloured values indicate exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 4.46. *Estimates of bystander exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)—1 day per year direct spray.*

Year	Ground-based exposure at 30 m (µg/kg/day) (MTO)				Aerial (fixed)-based exposure at 500 m (µg/kg/day) (MNR)			
	2,4,5-T	TCDD			2,4,5-T	TCDD		
		Low[a]	Central	High		Low	Central	High
1953	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04[b]
1954	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1955	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1956	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1957	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1958	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1959	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1960	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1961	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1962	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1963	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1964	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1965	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1966	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1967	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1968	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1969	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1970	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1971	2.12E+00	1.41E-06	1.41E-06	1.41E-06	5.26E+00	3.49E-06	3.49E-06	3.49E-06
1972	2.12E+00	1.41E-06	1.41E-06	1.41E-06	5.26E+00	3.49E-06	3.49E-06	3.49E-06
1973	2.12E+00	1.41E-06	1.41E-06	1.41E-06	5.26E+00	3.49E-06	3.49E-06	3.49E-06

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[b] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

4.9. Exposure assessment uncertainties

Re-creating exposure events that occurred decades ago is rife with unknowns and uncertainties. The quantitative (or numerical) exposure assessment required the input of many assumptions involving large amounts of data and numerical variables. Some of these input variables were found in the scientific literature, while other information was scenario specific and obtained from various sources available to the panel. The goal of quantitative exposure assessment is to produce a conservative model to ensure that exposures are never underestimated. Given the tendency for the assumptions described below to overestimate exposure, the exposure assessment likely errs on the side of conservatism (safety).

Re-creating exposure events that occurred decades ago is rife with unknowns and uncertainties. The quantitative (or numerical) exposure assessment required the input of many assumptions involving large amounts of data and numerical variables. Given the tendency for the assumptions described below to overestimate exposure, the exposure assessment likely errs on the side of conservatism (safety).

Table 4.47 summarizes the assumptions incorporated into the exposure assessment, as well as the potential effects of these assumptions and associated uncertainties in the panel's conclusions. When possible, the panel erred on the side of caution and attempted to overpredict exposure.

Table 4.47. *Major uncertainties in the assessment of exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Exposure assumption	Justification	Effect on exposure assessment
Exposure scenarios	The exposure scenarios developed for the assessment are based on a reconstruction of hypothetical events. The degree of uncertainty associated with typical operational practices during the assessment period (e.g., the use of personal protective equipment, the manner in which herbicide products were handled and transported, etc.) is considered very high. As a result, a range of plausible exposure parameters (e.g., amount of protective clothing worn) was used to try to capture these uncertainties.	Neutral
Herbicide use	Patterns and amounts of herbicide use in Ontario were not clearly documented, so use patterns and amounts were based on whatever historical information was available. When data were not available, average uses were assumed.	Neutral
Spray conditions	Weather conditions and other spray variables for specific spray events were not known. Applicators were assumed to have followed documented procedures for spray activities (i.e., weather conditions, vehicle speed, nozzle setup).	Neutral
Personal protective equipment use	Ontario government agencies provided workers involved in herbicide spraying with clear directions for using personal protective equipment. Actual use of personal protective equipment is not known; low, central, and high estimates of protection were assumed for all scenarios. The central estimate was assumed to represent the most likely scenario.	Neutral
Application rates	The data available for herbicide application rates in Ontario were inconsistent. Specific rates were used when available; when not available, average rates and/or intended rates were used.	Neutral
Application rates	For all exposure estimates, application rates assumed the application of 2,4,5-T alone. However, most applications involved a mix of 2,4,5-T and 2,4-dichlorophenoxyacetic acid (2,4-D) and/or other herbicides/adjuvants. Thus, actual application rates would have been half of what was assumed.	Overestimate
Provincial workers	The number of people involved in the spraying was relatively small. Available documentation for the Ministry of Transportation[a] showed that each district used two teams of two herbicide applicators, with many workers continuing in the same role year after year (two-man teams; consistent throughout the year and year to year; two teams per district; 18 districts; maximum 72 workers per year [driver/sprayer]). Similar teams and participants were assumed for Ontario Hydro. Better documentation was available for the Ministry of Natural Resources.	Neutral
Contaminant levels	The 2,4,5-T used in Ontario contained manufacturing impurities (especially TCDD). For use before regulation, the panel based contaminant level estimates on information available for 2,4,5-T stocks used in Vietnam. For use after regulation, the panel assumed that contaminant levels fell within those deemed acceptable through the pesticide registration process at the time of use. No information was available for actual 2,4,5-T use in Ontario.	Overestimate

Exposure assumption	Justification	Effect on exposure assessment
Environmental media concentration	Data for 2,4,5-T and TCDD levels in environmental media in Ontario during the time of use were not available. More recent and present-day levels were not considered relevant for this assessment. Thus, media concentrations were estimated based on standard methods and assumptions and information available on application rates, contaminant levels, and application methods. Where possible, assumptions were selected to err on the side of caution.	Overestimate
Receptor characterization	Receptors parameters were characterized based on information provided by Health Canada and the Ministry of the Environment. These parameters are a blend of average receptor characteristics and upper bound tendencies.	Overestimate
Occupation exposure parameters	The panel estimated exposure for each occupational receptor using the U.S. EPA Occupational Pesticide Handler Unit Exposure Surrogate Reference Table and Pesticide Handlers Exposure Database. These exposure factors are based on dosimeter studies and are intended as generic factors. A range of factors is provided to characterize the type of personal protective equipment the occupational receptors used. Low, central, and high exposure estimates were used to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate was meant to capture the most realistic estimate.	Neutral
Time activity patterns	Specific information regarding receptor time activity patterns was not available. Standard assumptions were used to capture upper bound tendencies.	Overestimate
Absorption rates and bioaccessibility factors	Soil bioaccessibility for 2,4,5-T and all oral and inhalation absorption factors were assumed to be 1.0 (the theoretical maximum).	Over estimate
Annual spray duration	Based on the available information, a six-month spray season was assumed for all areas of Ontario, which is likely an underestimate for southern Ontario.	Neutral
Fish and wildlife exposure estimates	Fish and wildlife concentration data were not available for the time or location of spray activities. Concentration data were estimated based on a series of assumptions. When possible, these assumptions were selected to err on the side of caution.	Overestimate
Bystander exposure estimates	Drift concentration data were not available for the time or location of spray activities. Exposure concentrations were estimated based on a series of assumptions and information from the scientific literature. When possible, these assumptions were selected to err on the side of caution.	Overestimate

[a] *In most cases, the names of organizations referred to in this report have changed several times during the more than three decades covering the period of use of 2,4,5-T in Ontario. The panel chose to refer to them by their most common or current designation.*

5. Development of standards and regulations for pesticide use in Ontario

5.1. Charge question

As part of its mandate, the Independent Fact-Finding Panel on Herbicide 2,4,5-T was asked to:

> *Review the preparation, application and storage of 2,4,5-T herbicide as well as provincial occupational health and safety, and laws, standards and workplace practices including the use of personal protection equipment and applicable training in place at that time* (Province of Ontario 2011).

5.2. History of the development of provincial and federal health and safety legislation and regulations

In response to a request from the Government of Ontario, this chapter provides an overview of the development of legislation, regulations, workplace standards, occupational health and safety guidelines, and standards and formal training of provincial government staff in the use of chemicals and pesticides during the years 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) was in use in Ontario. Information in this chapter was extracted from legislation, regulations, and various documents of provincial government staff and reports in the central repository. Much of the information is generic—related to chemicals or pesticides in general—and is not specifically directed at or related to 2,4,5-T. However, 2,4,5-T would have fallen under this legislative/regulatory/operational framework during the time it was used in Ontario. The panel could not determine the effectiveness of these workplace standards or the extent to which they were followed or enforced in the field.

The names of organizations referred to in this report have changed several times during the more than three decades of 2,4,5-T use in Ontario. The panel chose to refer to them by their most common or current designation.

In Canada, Health Canada's Pest Management Regulatory Agency (PMRA) must register pesticides before they can be imported, manufactured, sold, or used. When the herbicides 2,4,5-T and 2 (2,4,5-trichlorophenoxy) propionic acid (2,4,5-TP) were used in this country, pesticide registration was administered by Agriculture Canada, with the involvement of several other federal departments.

Under the federal Constitution Act (1867), provinces and territories can create legislation related to property, civil rights, and local interests (s 92 (13), (16)), including to restrict or prohibit the use of products registered under federal pesticide legislation in their jurisdictions. Provincial legislation or regulations can be **more but not less** restrictive than federal. For example, provinces and territories may require pesticide use permits and impose requirements to regulate selling, using, storing, disposing of, and transporting pesticides, including training, certifying, and licensing pesticide applicators and vendors.

When phenoxy herbicides were first used in North America starting in the late 1940s, they were unregulated. From then until the 1970s, regulations focused on minimizing drift and damage to neighbouring crops rather than protecting the health and safety of applicators and bystanders.

The federal Weed Control Act of 1937 dealt with the responsibilities of weed control inspectors and the control of noxious weeds, but phenoxy herbicides such as 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-T did not become commercially available for about 10 years after that (Dominion of Canada 1937).

Federally, pesticides were first regulated under the Agricultural Pest Control Products Act (Dominion of Canada 1927). As of 1939, the act was cited as the Pest Control Products Act (PCPA; Dominion of Canada 1939).

In 1949, all regulations were repealed and replaced, with the definition of *pest* also including plant life. Exemptions included "any product that is prepared and used for the purpose of controlling pests by a person engaged in the business or occupation of pest control operator" (Pest Control Products Act, RSC 1949, s 16 (3)). Labels had to include "The name of each active substance and the percentage thereof, and the number representing a coefficient, viscosity, specific gravity or other factor shall be stated immediately following and on the same line as the word 'Guarantee'" (Pest Control Products Act, RSC 1949, s 16 (3)).

Phenoxy herbicides were first used in North America starting in the late 1940s. From then until the 1970s, regulations focused on minimizing drift and damage to neighbouring crops rather than protecting the health and safety of applicators and bystanders.

The act also specified the required form for printing and marking packages, tags, and labels (Pest Control Products Act, RSC 1949, s 16 (5)). Schedule B chemicals listed included 2,4-D but not 2,4,5-T. In 1952, penalties (via regulation under the PCPA) for misrepresentation were a maximum of $100 for the first offence, between $100 to $200 for a second offence, and $200 to $500 for subsequent violations (Pest Control Products Act, RSC 1952, c 209, s 17).

In 1953, under permissive legislation in the provincial Public Health Act of 1950, an Ontario Order-In-Council approved comprehensive regulations for using extermination substances prescribed by regulations. Prior to 1954, few extermination substances were regulated. The Public Health Act was designed to suppress disease; as pesticide use was considered more of a safety problem, the Ministry of Health's (MOH) legal branch felt that any legislation related to pesticides should be separate from the Public Health Act (A0141559, Page 6) .[1]

On December 2, 1954, the Government of Canada repealed and replaced all regulations under the PCPA (Pest Control Products Regulations under the Pest Control Products Act RSC 1952, c 209). The new version of the act included no major changes to pesticide label requirements. Schedule A chemicals included both 2,4-D and 2,4,5-T. In both versions of the act, chemicals appear to have been classified by the nature of the guarantee required on the label.

In 1956, the Provincial Pesticides Act was introduced and gave the province the power to regulate exterminator licensing (Pesticides Act, RSO 1956, c 63, s 10). O Reg 174/56 laid out groups of pesticides (Group A, B, and C substances). Descriptions of some of the regulations under this act are as follows:

- Approved in 1956, O Reg 174 defined four licence classes and three assistant license classes with each class restricted to using pesticides in specific groups. The provincial medical officer of health reviewed license applications, with annual renewals required. Licences were not granted unless the applicant had held an assistant exterminator's license for at least one year or had evidence of experience that the director of MOH's Division of Industrial Hygiene Health deemed equivalent to service as an assistant.

[1] Citations in the format *A0* refer to records that are contained within the MNR searchable database. These records are available to the public, subject to Canadian copyright provisions and the Freedom of Information and Protection of Privacy Act. To access these records, visit www.ontario.ca/245T.

- O Reg174/56 required examinations for potential licensees.
- O Reg174/56 also laid out steps to protect pesticide users, defining *protective clothing* to mean "rubber gloves, rubber footwear, a gas mask capable of absorbing any poisonous gases or dusts present, and clothing and headgear sufficient to leave a minimum of skin or hair exposed."
- The Pesticides Act (RSO 1956, c 63) defined *extermination* as "the destruction or control of insects, vermin, birds, rodents or other pests, fungi or vegetation in a building or vehicle or on land by the use of any substance prescribed by the regulations." However, while the act granted power to regulate, it included no specific herbicide licensing requirements or lists of herbicides.
- In 1958, Ontario added a Class 5 license for airborne application of Class B substances as well as a corresponding assistant's license (O Reg 194/58, s 1).
- In 1960, Ontario added a Class 6 license and corresponding assistant's license for the control of insects, fungi, or vegetation on land (O Reg 25/60, s 1).

In 1963, the Pesticides Act was amended (An Act to Amend the Pesticides Act, RSO 1963, c 104). The original 1956 definition of *extermination* was "the destruction or control of insects, vermin, birds, rodents or other pests, fungi or vegetation in a building or vehicle or on land by the use of any substance prescribed by the regulations" (Pesticides Act, RSO 1956, c 63, s 1 (b)). However, the substances listed for regulation did not include any herbicides.

After 1963, the definition of *extermination* included "destruction or control of insects, vermin, birds, rodents or other pests, fungi or vegetation in a building or vehicle or on land by the use of any toxic or noxious substance" (An Act to Amend the Pesticides Act RSO 1963, c 104, s 1).

According to Record A0141559, the Bill 53 amendment to the Pesticides Act brought herbicides under regulation for the first time. The definition of *herbicide* used in O Reg 5/64 was "any substance used for the destruction or control of vegetation." Group C substances were defined as "made up of all herbicides except methyl bromide" (O Reg 5/64, s 2 (c); Table 5.1).

Table 5.1. *Pesticide groups and exterminator classes in Ontario under O Reg 5/64.*

Item	Substance authorized for use in an extermination	Conditions for use	Exterminator class	Number of form of licence
1	Group A and Group B	Any use	Class 1	1
	Group B	Any use	Class 2	2
	Group C[a]	Any use	Class 3	3
4	Group A, B, or C[a]	Only in building, vehicle or on land occupied by himself or his employer	Class 4	4
5	Group B	Only from an airborne machine	Class 5	5
6	Group B	Only for control of insects or fungi on land	Class 6	6

[a]*Group C would have included 2,4,5-trichlorophenoxyacetic acid (2,4,5-T).*

In 1967, the Government of Ontario created the Pesticide Advisory Board: "The Lieutenant Governor in Council may appoint a board consisting of nine members to be known as the Pesticides Advisory Board" (Pesticides Act,

RSO 1967, c 74, s 5). The board was responsible for examining license applicants, acting as an appeal board, and performing related functions.

In 1967, O Reg 445/67 was introduced under the Pesticides Act. This regulation changed the pesticide classification system to four groups (A, B, C, and D) and provided definitions for herbicides and hormone-type herbicides. Group D covered all herbicides except methyl bromide (O Reg 445/67, s 21, s 1(b), s 1(c)). This regulation created the licence classes described in Table 5.2 for land exterminations.

Table 5.2. *Land exterminator's licence classes in Ontario, as introduced under O Reg 445/67.*

Class	Authorized substances	Conditions of use
Class 1	Group A	Outside a structure
Class 2	Group B and C	On land not used in conjunction with plant and animal production
Class 3	Group B, C, and D[a]	From an airborne machine
Class 4	Group B and C	Concentrated air blast machine or power duster
Class 5	Group C	On land used for plant and animal production
Class 6	Group D[a]	On land not used in conjunction with plant or animal production
Class 7	Group D[a]	On his own premises or premises of his employer
Class 8	Group C	On his own premises or premises of his employer
Class 9	Group D[a]	Concentrated air blast machine

[a]*Group D would have included 2,4,5-trichlorophenoxyacetic acid (2,4,5-T).*

In 1967, according to an internal provincial government memorandum, an annual operator's licence was introduced for provincial government employees: "Under the Pesticides Act revised May 1967 government agencies are no longer exempt from regulations requiring the licensing of exterminators. Any person applying herbicides and including supervisors responsible for the application of herbicidal chemicals must now hold a license as prescribed in the Act" (A0134709, Page 2).

Also in 1967, Regulation 445 expanded licensing requirements to include reference letters (O Reg 445/67, s 8).

Under the 1967 Pesticides Act, O Reg 445/67 required that herbicides must not come into contact with areas other than those to be treated (O Reg 445/67, s 23) and that no substance should be stored in a manner that it is likely to come into contact with food or drink for human consumption (or animal) (O Reg 445/67, s 27). It also required that:

> *An empty container that has been used to hold a substance used in an extermination shall be disposed of,*
>
> *(a) in the case of combustible containers, by burning in such a manner that no one is exposed to the smoke;*
>
> *(b) in the case of a metal or glass container, by puncturing or breaking and burying the container in such a manner that it is covered by at least eighteen inches of earth and it does not pollute any water course or table* (O Reg 445/67, s 29)

Finally, it stated: "No operator or exterminator shall have in his possession a substance for extermination in a container other than the container in which it was originally offered for sale" (O Reg 445/67, s 28, (i)).

These standards from O Reg 445/67 were carried forward to the Pesticides Act, O Reg 657/70 (sections 27, 31, 33, and 32, respectively).

In 1967, before aerially applying herbicides (including 2,4,5-T), the licensed exterminator had to notify and get the consent of the director, meaning "an officer of the Department designated by the Minister as Director for the purpose of this Regulation" (O Reg 445/67, s 71).

In 1969, the licensing system was amended to add five more exterminator's licence classes to the existing nine (total of 14 licences, seven of which were based on Group D substances–for herbicides) as outlined in Table 5.3 (O Reg 139/69, s 9).

Table 5.3. *Land exterminator's licence classes in Ontario, as amended in 1969 (O Reg 139/69, s 9).*

Class	Authorized substances	Conditions of use
Class 1	Group A	Outside a structure
Class 2	Group A	On his own premises or premises of his employer
Class 3	Group B and C	On land not used in conjunction with plant and animal production
Class 4	Group B and C	Concentrated air blast machine or power duster
Class 5	Group C	On land used for plant or animal production
Class 6	Group C	On his own premises or premises of his employer
Class 7	Group B and C	From an airborne machine
Class 8	Group D[a]	From an airborne machine
Class 9	Group D[a]	For horticultural maintenance
Class 10	Group D[a]	On public roads
Class 11	Group D[a]	On rights of way
Class 12	Group D[a]	On industrial premises
Class 13	Group D[a]	On aquatic vegetation
Class 14	Group D[a]	Via concentrated air blast machine

[a]Group D would have included 2,4,5-trichlorophenoxyacetic acid (2,4,5-T).

In 1969, O Reg 139/69 was introduced under the Pesticides Act. This regulation limited attempts to pass a license exam to no more than twice in any 12-month period (O Reg 139/69, s 2).

In 1970, under the Pesticides Act, the Pesticide Advisory Board was dissolved, and the Pesticides Licence Review Board (PLRB) and Ontario Pesticides Advisory Committee (OPAC) were created (An Act to Amend the Pesticides Act 1967, RSO 1970, c 104, s 1). OPAC's role was to advise the Minister of Environment on pesticide-related matters, including:

- annually reviewing the workings of the Pesticides Act and its regulations
- reviewing government publications related to pesticides
- overseeing pesticide research funded by the ministry
- classifying all registered pesticides into schedules
- investigating areas of concern related to pesticides (A0148483, Page 2)

The Lieutenant Governor in Council was given the authority to appoint OPAC members (An Act to amend the Pesticides Act 1967, RSO 1970, c 104, s 2). The members had scientific backgrounds, and most were from outside of government.

In 1970, the Pesticides Act was changed to allow the Minister of Health, with the approval of the Lieutenant Governor in Council, to make regulations (Pesticides Act RSO 1970, c 346):

- "governing, regulating or prohibiting the use, handling or storage of substances used for extermination" (s 21 (19))
- "governing the storage and disposal of any unused portion of any substance used for extermination" (s 21(20))
- "requiring persons who handle or use any designated substance used for extermination to undergo medical examination and supervision, and providing for such medical examination and supervision" (s 21(23); extermination was defined as both land and structural extermination (s 1(e))

In 1970, the Ontario Minister of Health appointed a task group to study and report on the classification, distribution, sale, storage, and transportation of pesticides in Ontario (A0172631). In 1972, the Report by the Committee Appointed to Study Classification, Transportation, Distribution, Storage and Sale of Pesticides in Ontario was submitted to the Minister of Environment. The report recommended that:

- All pesticides should be legally classified into four categories:
  - A (restricted)
  - B (commercial)
  - C (home and garden)
  - D (unrestricted)
- Pesticide vendors should be subject to regulations limiting storage, transportation, and sale of pesticides.
- OPAC should classify all pesticide products marketed in Ontario (about 4,500) (A0172631, Page 7).

Note: In Table 1 of the report, all products containing 2,4,5-T amines or 2,4,5-T esters were listed as Category B (Commercial) (A0172631, Page 17).

In 1971, the Environmental Protection Act (EPA) was enacted. Part VI (Herbicides and Pesticides, s 54(1)) provided that any order, licence, permit, or regulation made under the Pesticides Act of 1967 continued to exist under the EPA until it was amended or revoked.

In 1972, the Ontario Ministry of the Environment (MOE) was created (Government Reorganization Act SO 1972, c 1, s 3(1)).

On November 25, 1972, the new federal Pest Control Products Act (PCPA) came into force (Pest Control Products Act RSC 1969).

In 1973, the province dissolved the PLRB and created the Pesticides Appeal Board (Pesticides Act RSO 1973, c 25). The new board provided a venue for applicants to appeal a director's decision to decline their license, with

director defined as "the officer of the Ministry designated by the Minister to perform the functions of the Director under this Act" (c 25, s 12-16).

In 1973, the Government of Ontario added the following section to Chapter 25 of the Pesticides Act:

> *Unless exempt by the regulations, no person shall sell, offer to sell or transfer any pesticide unless the pesticide is classified by the regulations and except under and in accordance with a licence that shall be for such class and in respect of each premises on, in or from which the pesticide is or will be sold, offered for sale or transferred* (Pesticides Act RSO 1973, c 25, s 5).

In 1973, the Government of Ontario added the following clause to Chapter 25 of the Pesticides Act: "This Act binds the Crown" (Pesticides Act RSO 1973, c 25, s 25).

In 1974, agriculturists performing land exterminations on farmland for agriculture or forestry purposes with schedule 2, 3, 4, or 5 pesticides were exempted from licence requirements (O Reg 618/74, s 73).

In 1974, Agriculture Canada published Trade Memorandum T-87, which refers specifically to the contaminant of 2,4,5-T known as 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). The memorandum said that 2,4,5-T products proposed for registration meet (among others) the following criteria: "a declared and certified TCDD content in the technical 2,4,5-T of less than 0.5 ppm" (A0137443, Page 1).

In 1975, the permissible TCDD content was further restricted. "The allowable TCDD content in technical material will be 0.1 ppm and analytical methodology will be required to demonstrate sensitivity to a level of 0.1 ppm TCDD" (A0137443, Page 1).

The reproductive toxicity of 2,4,5-T was first reported in 1969, after Bionetics Research Laboratory (under contract with the National Cancer Institute) completed a study entitled Evaluation of Carcinogenic, Teratogenic, and Mutagenic Activities of Selected Pesticides and Industrial Chemicals. Results of studies of rats administered high doses of 2,4,5-T revealed teratogenic and fetotoxic effects (IOM 1994).

During the early 1970s, TCDD was identified as a contaminant in commercial products of 2,4,5-T and 2,4,5-TP registered in Canada. As provincial legislation and regulations can be more—but not less—restrictive than federal ones, the new federal requirement for TCDD content had an immediate effect in Ontario.

During the early 1970s, TCDD was identified as a contaminant in commercial products of 2,4,5-T and 2,4,5-TP registered in Canada. As provincial legislation and regulations can be more—but not less—restrictive than federal ones, the federally imposed limits on TCDD levels in 2,4,5-T had an immediate effect in Ontario.

In 1974, O Reg 618/74 was introduced under the Pesticides Act of 1973, replacing O Reg 657/70. The four groups of pesticides (A, B, C, and D) were replaced with five schedules. The schedules listed the herbicides (by their trade names) and were included with the regulation. However, the regulation did not define the criteria used to place products into the five schedules. Products containing 2,4,5-T were classified into schedules 1, 2, or 3.

Regulation 618/74 listed ten classes of licences (Table 5.4).

The schedules were listed in the regulation and updated periodically via an amending regulation (O Reg 618/74).

Licensed exterminators who wanted to aerially apply 2,4,5-T or 2,4,5-TP (and any other plant growth-regulating substances) had to get a use permit from the director (O Reg 618/74, s 66(1)). O Reg 618/74 also exempted weed inspectors from licence requirements (s 76).

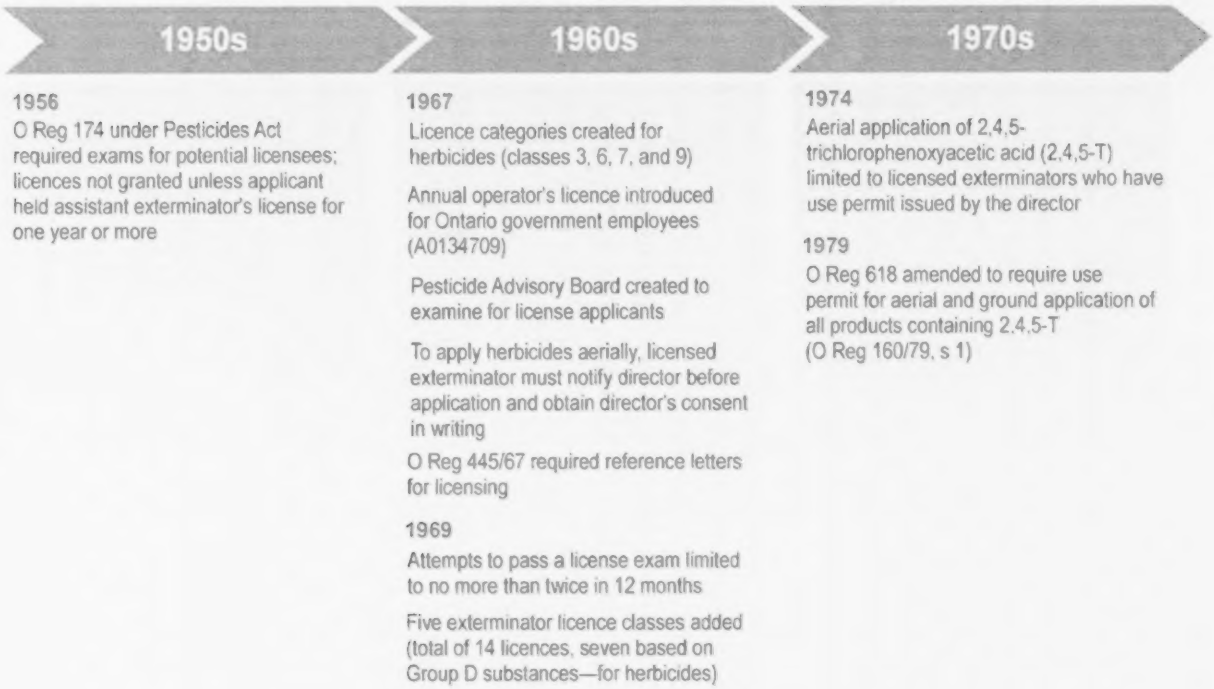
Table 5.4. *License classes in Ontario, as outlined under O Reg 618/74, s 59.*

Class	Authorized substances	Conditions of use
Class 1	Schedule 2, 3, and 4 pesticides that are herbicides[a]	Non-agricultural use
Class 2	Schedule 3 and 4 pesticides that are herbicides[a]	Non-agricultural use
Class 3	Schedule 2, 3, and 4 pesticides other than herbicides	Non-agricultural use
Class 4	Schedule 3 and 4 pesticides other than herbicides	Non-agricultural use
Class 5	Schedule 2, 3, 4, and 5 pesticides other than herbicides	Agricultural land
Class 6	Schedule 2, 3, and 4 pesticides that are herbicides[a]	Agricultural land
Class 7	Schedule 2, 3, 4, and 5 pesticides other than herbicides	From an airborne machine
Class 8	Schedule 2, 3, and 4 pesticides that are herbicides[a]	From an airborne machine
Class 9	Schedule 2, 3, 4, and 5 pesticides other than herbicides	Concentrated air blast machine and power duster
Class 10	Pesticide(s) stipulated on licence	Use, premises, and/or equipment stipulated on licence

[a]*Would have included products containing 2,4,5-trichlorophenoxyacetic acid.*

Figure 5.1 presents dates for key legislative licensing requirements in Ontario.

Figure 5.1. *Key dates related to pesticide licensing requirements in Ontario.*


1950s
1956
O Reg 174 under Pesticides Act required exams for potential licensees; licences not granted unless applicant held assistant exterminator's license for one year or more
1960s
1967
Licence categories created for herbicides (classes 3, 6, 7, and 9)
Annual operator's licence introduced for Ontario government employees (A0134709)
Pesticide Advisory Board created to examine for license applicants
To apply herbicides aerially, licensed exterminator must notify director before application and obtain director's consent in writing
O Reg 445/67 required reference letters for licensing
1969
Attempts to pass a license exam limited to no more than twice in 12 months
Five exterminator licence classes added (total of 14 licences, seven based on Group D substances—for herbicides)
1970s
1974
Aerial application of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) limited to licensed exterminators who have use permit issued by the director
1979
O Reg 618 amended to require use permit for aerial and ground application of all products containing 2,4,5-T (O Reg 160/79, s 1)

O Reg 618/74 under the Pesticides Act required that holders of vendor wholesale licences store pesticides (including 2,4,5-T) as follows:

> *(a) in such manner that the pesticide is not likely to contaminate food or drink intended for human or animal consumption;*
>
> *(b) in such a manner that the pesticide is not likely to impair the health or safety of any person;*
>
> *(c) in an area that is maintained in a clean and orderly manner and with precautions taken sufficient to prevent the pesticide from contaminating any other pesticide stored in the same area, or the natural environment; and*
>
> *(d) in an area that has a warning sign prominently displayed at the entrances thereof indicating the presence of a pesticide* (O Reg 618/74, s 100(1)).

The regulation laid out the following requirements for storage areas:

> *(a) that has no floor drain that leads into or drains directly or indirectly into a storm sewer, sanitary sewer or watercourse; and*
>
> *(b) near which the following articles which are kept by the licensee for emergency purposes and are readily available for such purposes,*
>
> *(i) a respiratory protection device of a type that will give protection from the pesticide,*
>
> *(ii) rubber boots,*
>
> *(iii) rubber gloves, and*
>
> *(iv) a hat and coat that will provide protection against any pesticide stored on the premises* (O Reg 618/74, s 100(2)).

In 1976, O Reg 577/76 was approved under the Pesticides Act of 1973 to include in the definitions:

> *(a) "adequate respiratory protection" means a respiratory device or devices which effectively protects the user from adverse effects which might result from breathing in of a pesticide during the handling or use of the pesticide;*
>
> *(aa) "adequate protective clothing" means clothing including rubber or neoprene boots, rubber or neoprene gloves, hats, coats and other garments that effectively protect the user from adverse effects which might result from a pesticide coming in contact with the skin during or after the handling or use of the pesticide* (O Reg 577/76, s 1).

O Reg 577/76 added a new schedule of pesticides, Schedule 6 (Table 5.5; O Reg 577/76, s 25).

Table 5.5. *Land exterminator's licence classes in Ontario, as outlined under O Reg 577/76, s 25.*

Class	Authorized substances	Conditions of use
Class 1	Schedule 2, 3, 4, and 6 pesticides that are herbicides[a]	Non-agricultural use
Class 2	Schedule 3, 4, and 6 pesticides that are herbicides[a]	Non-agricultural use
Class 3	Schedule 2, 3, 4, and 6 pesticides other than herbicides	Non-agricultural use
Class 4	Schedule 3, 4, and 6 pesticides other than herbicides	Non-agricultural use
Class 5	Schedule 2, 3, 4, 5, and 6 pesticides other than herbicides	Agricultural land
Class 6	Schedule 2, 3, 4, and 6 pesticides that are herbicides[a]	Agricultural land
Class 7	Schedule 2, 3, 4, 5, and 6 pesticides other than herbicides	From an airborne machine
Class 8	Schedule 2, 3, 4, and 6 pesticides that are herbicides[a]	From an airborne machine
Class 9	Schedule 2, 3, 4, 5, and 6 pesticides other than herbicides	Air-blast machines and power dusters
Class 10	Pesticide(s) stipulated on licence	Use, premises and/or equipment stipulated on licence

[a]*Would have included products containing 2,4,5-trichlorophenoxyacetic acid (2,4,5-T).*

In 1978, the Ontario Health and Safety Act (OHSA) was enacted and included language defining *designated substance* as a "biological, chemical or physical agent or combination thereof prescribed as a designated substance to which the exposure of a worker is prohibited, regulated, restricted, limited or controlled" (OHSA RSO 1978, c 83, s 1(6)). At the time, herbicides were regulated under the Pesticides Act and should have been considered designated substances.

However, the OHSA regulated toxic substances, and Section 20, Subsection 11 states, "This section does not apply to designated substances" (OHSA, RSO 1978, c 83, s 20(11)). It appears that the Ontario Ministry of Labour (MOL) clarified how the OHSA would interact with the Pesticides Act by including this *designated substances* definition and exemption, thereby avoiding covering herbicides under both acts. However, general provisions of the OHSA, such as employees having the right to reject unsafe work or supervisors being responsible for the conduct of employees, would still apply.

In 1978, under O Reg 575/78, the depth for disposal of containers of schedule 1, 2, or 5 pesticides was changed from 18 inches to 50 cm (18 inches = 45.72 cm so this change was likely to align with the metric system).

In 1979, the United States Environmental Protection Agency (U.S. EPA), under an emergency suspension order, temporarily banned the use of products containing 2,4,5-T and 2,4,5-TP based on a study that suggested a link between forestry use and miscarriages in Oregon (A0137137).

Also in 1979, British Columbia banned 2,4,5-T and restricted use of 2,4,5-TP (A0130973).

On March 12, 1979, the Ontario Minister of Environment made a statement in the Legislature that he had arranged "to pass an immediate regulation to the Pesticides Act that will temporarily prohibit the use of" 2,4,5-T and 2,4,5-TP until MOE staff and OPAC had time to review data on these herbicides (A0130969, Page 2).

Agriculture Canada and Health and Welfare Canada provided a memorandum of information to the Canadian Association of Pesticide Control Officers that outlined the U.S. EPA announcement for an immediate halt to sale

of 2,4,5-T and 2,4,5-TP. The memorandum also said that the Canadian government was monitoring developments but had decided not to take any regulatory action because reports prompting the U.S. EPA decision appeared inadequate to support any such action (A0130963, Page 2).

In a statement to the Legislature, the Ontario Minister of Environment said he had received an OPAC report that stated that the scientific basis for the U.S. study was inadequate and did not justify the U.S. EPA action (A0148474, pages 15-20).

In 1979, O Reg 618/74, s 61(3) was amended to require a use permit for aerial and ground application of all products containing 2,4,5-T. Previously, only **aerial** application of 2,4,5-T or 2,4,5-TP required a use permit (O Reg 160/79, s 1).

At the same time, the Minister of Environment directed that no permits could be issued until the ministry and OPAC assessed relevant U.S. information, which was then held in legal action.

OPAC recommended that 2,4,5-T and 2,4,5-TP remain available for use subject to permit conditions. However, the Minister of Environment indicated he was reluctant to act on OPAC's recommendation, due to great public concern; thus, in Ontario permits for using these herbicides continued to be denied. The Minister also stated that he had received an OPAC report on Ontario government and agencies' use of 2,4-D, other phenoxy herbicides, and picloram (A0134305, Page 3).

In 1979, the Pesticides Appeal Board was dissolved and the Environmental Appeal Board created (Pesticides Act RSO 1979, c 79 s 1).

In 1980, the MOE advised all holders of 2,4,5-T and 2,4,5-TP products that they must retain all stock until a safe way to destroy it could be found (A0138370, pages 3-4).

Even as late as 1980, O Reg 751/80 under the Pesticides Act defined *hormone type herbicide* and specifically included 2,4,5-T (O Reg 751/80, s 1(m)(iii)). In 1981, 2,4,5-T was still listed in the schedules (for example, O Reg 756/81, Schedule 1, Page 1637; Schedule 2, Page 1640).

In 1979, following the U.S. EPA announcement of the immediate stop sale of 2,4,5-T and 2,4,5-TP, Agriculture Canada and Health and Welfare Canada announced that the Canadian government had decided not to take any regulatory action because it appeared that the data on which the U.S. decision was based was inadequate to support such action (A0130963, Page 2).

Similarly, in 1979, the Ontario Minister of Environment was advised by the Ontario Pesticide Advisory Committee (OPAC) that the scientific basis for the U.S. study was inadequate and did not justify the U.S. EPA action (A0148474, pages 15-20).

Also in 1979, pursuant to O Reg 618/74, s 61(3), the Ontario Minister of Environment directed that no permits would be issued for aerial and ground application of all products containing 2,4,5-T until the ministry and OPAC had assessed the information on which the U.S. EPA stop sale order had been based.

The Government of Canada did not renew the registration of 2,4,5-T after December 31, 1985. By that time, the chemical was no longer available in Canada.

Figure 5.2 presents key legislative and regulatory dates related to 2,4,5-T use in Ontario.

Figure 5.2. *Key legislative/regulatory dates for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) use in Ontario.*

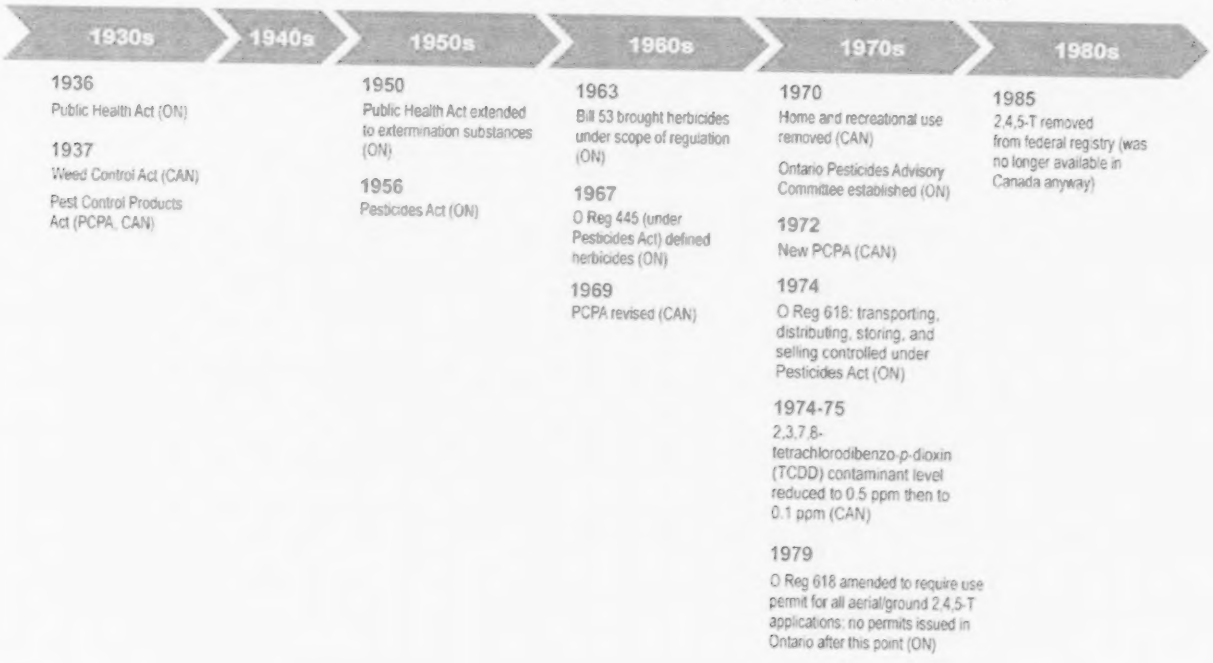
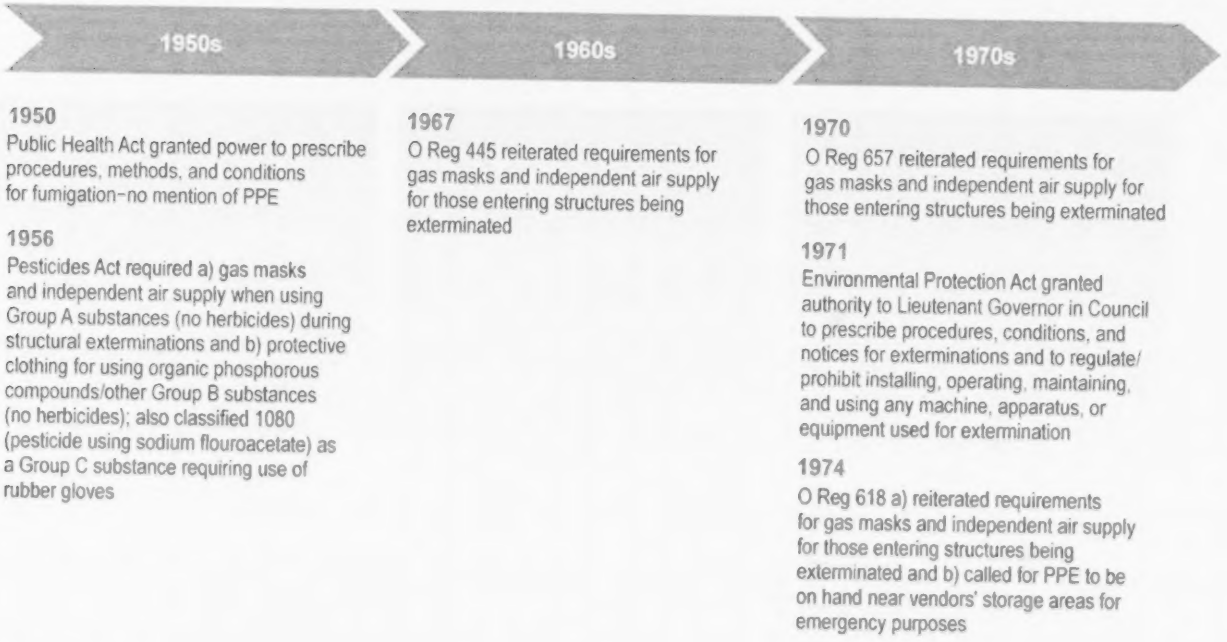

1930s
1940s
1950s
1960s
1970s
1980s
1936
Public Health Act (ON)
1937
Weed Control Act (CAN)
Pest Control Products Act (PCPA, CAN)
1950
Public Health Act extended to extermination substances (ON)
1956
Pesticides Act (ON)
1963
Bill 53 brought herbicides under scope of regulation (ON)
1967
O Reg 445 (under Pesticides Act) defined herbicides (ON)
1969
PCPA revised (CAN)
1970
Home and recreational use removed (CAN)
Ontario Pesticides Advisory Committee established (ON)
1972
New PCPA (CAN)
1974
O Reg 618: transporting, distributing, storing, and selling controlled under Pesticides Act (ON)
1974-75
2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) contaminant level reduced to 0.5 ppm then to 0.1 ppm (CAN)
1979
O Reg 618 amended to require use permit for all aerial/ground 2,4,5-T applications; no permits issued in Ontario after this point (ON)
1985
2,4,5-T removed from federal registry (was no longer available in Canada anyway)

Figure 5.3 presents key legislative and regulatory dates related to personal protective equipment in Ontario.

Figure 5.3. *Key legislative/regulatory dates for personal protective equipment (PPE) in Ontario.*


1950s
1960s
1970s
1950
Public Health Act granted power to prescribe procedures, methods, and conditions for fumigation–no mention of PPE
1956
Pesticides Act required a) gas masks and independent air supply when using Group A substances (no herbicides) during structural exterminations and b) protective clothing for using organic phosphorous compounds/other Group B substances (no herbicides); also classified 1080 (pesticide using sodium flouroacetate) as a Group C substance requiring use of rubber gloves
1967
O Reg 445 reiterated requirements for gas masks and independent air supply for those entering structures being exterminated
1970
O Reg 657 reiterated requirements for gas masks and independent air supply for those entering structures being exterminated
1971
Environmental Protection Act granted authority to Lieutenant Governor in Council to prescribe procedures, conditions, and notices for exterminations and to regulate/prohibit installing, operating, maintaining, and using any machine, apparatus, or equipment used for extermination
1974
O Reg 618 a) reiterated requirements for gas masks and independent air supply for those entering structures being exterminated and b) called for PPE to be on hand near vendors' storage areas for emergency purposes

5.3. Standard operating procedures for preparing, loading, mixing, and applying herbicides

Ontario government agencies first produced standard operating procedures for preparing, loading, mixing, and applying herbicides during the 1950s. In some cases, particularly with the Ministry of Natural Resources (MNR), standard operating procedures appear to have evolved over time and to have followed established field operational practices.

Ontario government agencies first produced standard operating procedures for preparing, loading, mixing, and applying herbicides during the 1950s. In some cases, particularly with MNR, standard operating procedures appear to have evolved over time and to have followed established field operational practices.

In 1954, Ontario Hydro produced the Manual of Spray Practices, which said that herbicides are known to be non-toxic to man and animals but operators should minimize breathing in the mist or having it contact their faces (A0138473, Page 69).

The manual also advised that workers should use coveralls and rubber boots and gloves for "the handling of amate" and that those with allergies should not work with spray materials (A0138473, Page 69). The main concerns for health and safety involved operators slipping/tripping due to oily surfaces or hoses or being burned or injured due to oil fires, hot exhaust pipes, or lifting of heavy containers. The manual also contained cautions related to drift, crop damage, and application in the wind (A0138473, pages 69-71). The 1958 Ontario Hydro Manual of Spray Practices (A0138491, Page 85) contained largely the same health and safety information as the 1954 version.

By 1962, the Ontario Hydro Manual of Spray Practices (A0141556) was expanded to include the following on personal safety:

- "Spraying should be done in such a manner as to prevent the chemical coming into contact with the eyes" (A0141556, Page 29).
- "Protective clothing should be worn. Numerous items are available through Central Stores, such as neoprene suits, gloves and boots, safety insoles and hard hats" (A0141556, Page 29).
- "Avoid letting chemical contact skin whenever possible" (A0141556, Page 177).

Most of the safety issues covered in the 1962 manual related to spray drift and hoses as tripping hazards: "Spray chemicals can, however, be harmful if used indiscriminately. Some people have been found to be allergic, with resultant eczema or rashes....All chemicals should therefore be handled with caution and reasonable care" (A0141556, Page 32).

In 1963, the Ontario Ministry of Agriculture, Food, and Rural Affairs (OMAFRA) published the first Guide to Chemical Weed Control (A0141583). A small section entitled Use Herbicides Safely remained the same in the 1964 (A0141584), 1976 (A0141606), and1980 editions (A0141620). This section advised:

- "Follow directions! Read the instructions and follow the precautions printed on the container labels" (A0141584, Page 10; A0141606, Page 10; A0141620, Page 13).
- "Protect the eyes and avoid inhaling chemicals or prolonged exposures of the skin to the chemical. Take special care when the herbicide is labeled "poisonous" (A0141584, Page 10; A0141606, Page 10; A0141620, Page 13).

Similar to the Ontario Hydro manuals of spray practices, OMAFRA guides to chemical weed control placed more emphasis on avoiding drift to susceptible crops than on applicators' personal safety. The latter guides also mentioned using a "special sprayer for herbicides such as 2,4,D or 2,4,5-T. These chemicals are often impossible to remove from a sprayer and traces of them in other solutions may damage susceptible plants" (A0141584, Page 10; A0141606, Page 10; A0141620, Page 13).

In 1965, the Ministry of Transportation (MTO) issued Circular 65-005, which listed neoprene gloves and blue coveralls as safety clothing available for weed spray crews (A0134563, Page 4), with apron and rubber boots also listed for "Distributor Spray Bar Operators" (A0134563, Page 5). The circular did not advise that this clothing was required: "An employee shall be responsible for cleaning and maintaining garments, and shall bear the cost thereof, while they are in his possession and on charge to him" (A0134563, Page 2).

In 1971, MTO issued Circular 71-038 (A0134562), which called for applicators to use the same equipment as specified in Circular 65-005 (A0134563).

Starting in the 1960s, manuals began to indicate that instructions and precautions printed in the pesticide label should be followed (label requirements were defined under the federal PCPA). Labels from the early 1970s included the following precautionary statements:

- May cause skin irritation
- Keep out of reach of children
- Do not take internally
- Avoid contact with skin, eyes or clothing (A0138949, A0138931, A0137453, A0137452, A0138340, A0132951, A0130983, A0130934)

By the mid- to late 1970s, standard operating procedures for Ontario government agencies were much more prescriptive about using personal protective equipment and using pesticides safely. Applicator health and safety became a higher priority. During the mid- to late 1970s, OMAFRA, MTO, and MOE produced pesticide manuals that included detailed safety instructions for applicators.

In 1976, OMAFRA produced Fruit Production Recommendations. The section entitled Safe Use of Pesticides advised "when measuring out pesticides: wear heavy rubber gloves, avoid splashing and spilling, do not inhale the dust when filling the sprayer or duster....Do not work in drenched clothing. Wash clothing before wearing it again" (A0156174, Page 5).

The 1976 MTO Pesticides Spray Manual advised that "each piece of equipment used in the application of a pesticide shall be operated only by personnel holding a valid exterminators license" (A0142281, Page 6). Pesticide safety practices were as follows:

> *Applicator Safety*
>
> *(i) Study and comply with instructions and precautions outlined on the container labels.*
>
> *(ii) Handle volatile materials in a well-ventilated area to avoid over-exposure to fumes.*
>
> *(iii) Avoid over-exposure to spray drift.*

(iv) Wear protective clothing such as coveralls, rubber gloves etc., when handling pesticides in their concentrated form.

(v) Should any spray materials spill on skin, hands, face or in eyes, wash immediately.

(vi) Wash clothing frequently when handling or applying pesticides, preferably every day.

(vii) Change clothing immediately should it become contaminated by spilled or splashed pesticides.

(viii) Do not smoke or eat while handling or applying pesticides. Wash hands and face thoroughly before eating or smoking.

(ix) If symptoms of illness occur during or shortly after spraying, obtain immediate medical attention, and notify immediate supervisor (A0142281, Page 7).

Safety in Application

(i) *Respect the potential hazards of chemicals and only use as recommended.*

(ii) Properly calibrate sprayers prior to commencement of spraying to ensure that the correct amount of material is applied.

(iii) Keep sprayers in top operating condition by repairing or replacing nozzles, gauges, valves etc., at the first indication of a malfunction.

(iv) Take every precaution to confine the spray pattern to the treatment area.

(v) Do not spray near crops, gardens, flowers, or ornamental shrubbery.

(vi) Do not wash out spray equipment in or near lakes, ponds, or streams or where runoff could affect potable water.

(vii) No pesticide is to be applied closer than 100 feet to any bodies of water such as lakes, rivers, streams, and wet areas that could be sources of potable water.

(viii) Do not apply any pesticide closer than 100 feet to built up areas, single dwellings, or recreational areas such as golf courses, parks, picnic areas, private campgrounds or any other area where people congregate for recreational purposes.

(ix) Sprays should be applied with the wind when possible and always downwind of susceptible crops.

(x) Stop all spraying when temperature is high enough to readily volatize the chemical involved. The safe upper limit for spraying has been determined to be approximately 32°C (90°F).

(xi) Stop all spraying when the wind is strong enough to carry spray particles in droplet form away from the area to be treated. Generally speaking, no spraying should be done with conventional water based sprays when the wind exceeds 10 M.P.H, and with thickened spray materials when the wind is in excess of 20 M.P.H., keeping in mind that these values are maximum limits when spraying with the wind (A014228, Page 8).

The 1976 MTO Pesticides Spray Manual (A0142281) dealt only with ground application (not aerial) and described daily temperature fluctuations and temperature conditions under which spraying is most effective. It also had detailed sections on how to calibrate and maintain equipment.

The 1977 MOE Chemical Safety Handbook (A0137711) advised what action to take in the event of a major crash or spill involving pesticides. The manual also presented first aid treatment if a pesticide splashed in eyes or on skin, saying:

- "Each person who is involved in the handling and distribution of pesticides must be vigorously and repeatedly impressed with the seriousness of allowing even a tiny amount of these chemicals to be taken into the body by breathing, swallowing, or absorption through the skin. It is wise to assume that all pesticides may be absorbed through any one of these three routes, and protective measures need to be strictly adhered to with this very much in mind" (A0137711, Page 9).
- "Furthermore in every instance of pesticide application someone has to assume responsibility for the conduct of the operations....A person should never be allowed to work alone under conditions in which he could not receive prompt help in the case of an accident" (A0137711, Page 10).
- "It is recommended that copies of the following page should be prominently displayed in every work area, aircraft, truck, storage area, waiting area, and washroom" (A0137711, pages 10-11).

This chemical safety handbook also provided the following rules for chemical safety:

Twelve Rules for Chemical Safety:

1. *Inspect pesticide container for leaks before handling them.*
2. *Do not handle containers roughly or carelessly.*
3. *Should a leak or spill occur, keep people and animals away from the area; decontaminate thoroughly and report immediately to the Supervisor of the Pesticide Control Section, Ministry of the Environment, 135 St. Clair Avenue West, Toronto M4V1P5. Telephone: (416) 965-2401.*
4. *Inspect vehicles for contamination after unloading; do not permit a contaminated vehicle to leave.*
5. *Do not store pesticides or empty pesticide containers anywhere near food or drink (including that for animals).*
6. *Do not keep food, drink, tobacco, cups, or cutlery anywhere in the work areas or in work clothes.*
7. *Do not eat, drink or smoke in a work area.*
8. *Do not rub the eyes or touch the mouth while working with pesticides.*
9. *Wash hands thoroughly before eating, drinking, smoking, or using the toilet.*
10. *Wear clean rubber gloves and protective clothing when handling pesticides, and a respirator whenever recommended.*
11. *Never fail to discard contaminated clothing or faulty protective covering, especially gloves.*
12. *Read the label carefully; if seeking medical aid take the label and/or container with you* (A0137711, pages 10-11).

The handbook also provided the following detailed list of protective clothing:

Protective Clothing and Respirators

Proper protective clothing and equipment should always be worn when handling, mixing or applying pesticides. A water repellent hat with a full brim, rubber boots, rubber gloves, coveralls, rubber apron should be worn to protect the skin from exposure to pesticides.

A respirator approved by the United States Bureau of Mines and/or the National Institute for Occupational Safety and Health (NIOSH) should always be worn whenever there is a risk of dust or vapour being inhaled. A respirator should cover at least the mouth and nose and must fit well. Cartridges or canisters should be changed about every 8 hours or sooner if an odour of the material is detected by the operator, once the canister becomes saturated, it no longer protects the user. A list of approved respirator devices for protection against pesticides can be obtained from the pesticide Control Specialist, Ministry of the Environment, in your area (A0137711, Page 39).

This chemical safety handbook also described these measures to prevent accidents:

Prevention

All personnel should train themselves to observe the following guidelines:

1. *Open all pesticide containers carefully, and on a stable surface where they won't tip or spill easily.*
2. *Open, pour and mix in a specific area where no person can be contaminated, and where any spills can be cleaned up properly.*
3. *Use the proper tools to open containers. Use a knife to open paper and plastic bags, and to cut tops. (Don't use a screwdriver or pickaxe, etc., because of the risk of material spurting out onto face and eyes). Ripping open a bag usually causes an uneven tear, thus making spills more likely.*
4. *Stand upwind of all opening, pouring and mixing operations.*
5. *Open, pour, weigh, and mix in a well-ventilated area.*
6. *Learn how to pour properly from a container. Splashing and spurting can be avoided if a can is held so that the opening is at the top. If an air vent is provided, use it.*
7. *Wear clean and well-fitted goggles or respirator, (or a face shield), rubber gloves, coveralls, rubber boots and apron for protection in the event of a splash or spill – especially when handling concentrated or highly toxic materials.*
8. *Be sure you know where to easily and quickly obtain a good supply of lime, coarse clay, sand, sawdust, or other absorbent to soak up a spilled pesticide.*
9. *Do not permit any person to work alone, especially when handling a highly toxic pesticide.*
10. *A pilot must never take part in mixing and loading operations, and should stand well away.*
11. *Learn to recognize the typical signs and symptoms of pesticide poisoning.*
12. *If you feel ill during pesticide application, or shortly after, stop work and seek medical attention at once. Do <u>not</u> carry on because of the work schedule.*
13. *Never use the mouth to siphon liquid materials or to blow out a clogged spray nozzle.*
14. *Keep all unprotected persons away from <u>any</u> equipment that may be contaminated. Consider all such equipment dangerous until properly decontaminated.*
15. *Remember that equipment used for the application of herbicides should be reserved exclusively for that purpose.*
16. *Make sure that personnel who maintain or clean equipment are aware of the hazards and follow safety procedures similar to those for handling the toxic products themselves. Do not permit anyone unfamiliar with chemical safety practices to carry out cleaning procedures.*
17. *Ensure that smoking, drinking and eating are absolutely prohibited in every chemical handling area.*

18. *Change clothes daily, and more often if any contamination occurs.*
19. *Shower thoroughly, with special attention to hair and fingernails, before going home.*
20. *Never fail to discard leaky gloves or contaminated leather boots or shoes.*
21. *Know the limitations of protective clothing and equipment, especially which respirator cartridge to use and how often to change it. Remember that each person's protective outfit must be complete.*
22. *Know that a very serious risk is taken if a victim of pesticides poisoning is allowed to drive home unattended. It is simply not possible to judge the ultimate severity by the initial symptoms. A severe poisoning may seem quite mild at the outset. Once poisoning is suspected, someone should stay with the patient until he reaches medical treatment, and he certainly should not operate a vehicle.*
23. *Be certain to read the label of every pesticide used. These are carefully prepared and carry the necessary information respecting rates, uses, cautions, and hazards. If a label is damaged or illegible, do not use that material. Be sure you know the chemical being used.*
24. *Make sure that the aircraft tank or hopper openings are securely closed.*
25. *Make sure that water, soap and towels are available at the loading site* (A0137711, pages 39-41).

Finally, this handbook included a section devoted to decontaminating equipment used to apply herbicides:

Decontamination of Application Equipment - Chlorophenoxy Compounds

After the use of herbicides such as 2,4-D or 2,4,5-T, it is necessary to remove the chemical completely from the system, to prevent contamination of the next charge of spray and thus damage to crops. However, complete decontamination is generally considered impossible for these compounds. If the same equipment must be used for the application of other chemicals, the following methods for 'cleaning' are offered:

For either soil or water soluble solutions:

i. *Add 1 ounce of activated charcoal and 1 ounce of detergent to each 2 gallons of water. Shake well and discharge through nozzles, or,*
ii. *Add 10 ounces of lye to 2 gallons of water and allow to stand at least 2 hours. Then discharge through the nozzles and rinse the system twice with clean water.*

For water-soluble solutions only:

iii. *Add 1½ ounces of washing soda to each 2 gallons of water and proceed as in ii.*
iv. *Add ½ cup of household ammonia to each 2 gallons of water. Flush some through the nozzles and leave the rest in the tank overnight. Then discharge and thoroughly rinse the system with water.*

For oil-soluble formulations only:

v. *Add 1½ cups of kerosene, 1½ ounces of washing soda, and a little detergent to each 2 gallons of water and proceed as in ii* (A0137711, pages 44-45).

In 1977, OPAC produced a report entitled Personal Protective Equipment for Pesticide Users (A0148465, Page 3). OPAC agreed that the report be submitted to the deputy minister for approval before the Pesticides Control Section printed and distributed it.

In 1978, the manager of the Ontario Hydro Department of Forestry sent a covering memo to Ontario Hydro forestry superintendents in all regions. The memo advised that:

In order to maintain a high level of safety performance ....remind those employees who use (handle, mix or apply) pesticide, of the precautions to minimize the chance of hazards of exposure to these chemicals:

1. *Read the container label for instructions re safe handling and use.*
2. *Use rubber or neoprene or equivalent gloves and suitable eye protection when working with the concentrated materials during mixing operations.*
3. *When applying the diluted mixture, avoid skin contact with the material by the use of:*
   (a) *Long sleeve shirts and full work pants when applying water based or dry materials. These should be laundered frequently (see Collective Agreement Item 25.0 (b) – Trades)*
   (b) *Approved, Corporation supplied protective clothing when applying oil based sprays.*
   (c) *Approved gloves shall be worn.*
4. *Avoid inhalation of airborne particulates by standing upwind, changing positions, etc.*
5. *After being exposed to pesticides, and before eating or smoking, employees should wash their hands thoroughly* (A0141711, Page 1).

Appended to the 1978 covering memo (A0141711, Page 1) was Forestry Department Directive 253 (supersedes Directive 70-1):

1. *Use only herbicides that are registered by Agriculture Canada, under the Pest Control Products Act, and is in accordance with the label specifications.*
2. *Comply with the Province of Ontario's Pesticides Act and Regulations with regard to the use, storage, handling and licensing requirements.*
3. *Conform to restrictions placed on the products by the manufacturer, and to uses and rates as set out in the Chemical Control of Brush, Weeds, Insects, Fungi and Stump Sprouting (Ontario Hydro Specification L-167). Department of Transmission Environment Technical Bulletins, Trade Reference Manuals and System Maintenance Directives.*
4. *Adhere to the restrictions under the agreement with the Ministry of Transportation and Communications on Joint Forestry Procedures.*
5. *Apply all herbicides selectively, ie, only to the target plants to be controlled wherever possible.*
6. *Have all Ontario Hydro herbicide applications supervised by provincially licensed personnel* (A0141711, pages 4-5).

By 1979, MNR had prepared its first draft of Operational Guide for Aerial Spraying. Although this manual was finalized after 2,4,5-T was no longer used in Ontario, it was a compilation of the various standard operating procedures that had been developed and refined over the previous three decades of aerial pesticide applications in the province. Thus, the panel included this guide in the review of standard operating procedures, as it summarizes the application procedures and safety measures that would have been in place while MNR was using 2,4,5-T.

In 1980, the supervisor of MNR's Pest Control Section sent a letter to MNR's northern regional supervisors to inform them of the Pest Control Section publication entitled Operational Guide for Aerial Spraying – First Draft 1979. The letter mentions that this manual was provided to "all participants of Aerial Application Seminar held in Kapuskasing last summer as well as to all attendees of the pesticide applicator training courses conducted by Pest Control Section this spring" (A0133107, Page 19).

In 1981, MNR published the final draft of Aerial Spraying for Forest Management. This manual said that a project supervisor:

> *(a) Should be located at the air base (mixing/loading site) during hours of operation*
>
> *(b) Responsible for :*
>
> *(i) overall control of the operation*
>
> *(ii) co-ordination of activities of all functions of the operation* (A0136248, Page 14).

The aerial spraying manual provided the following information on safety:

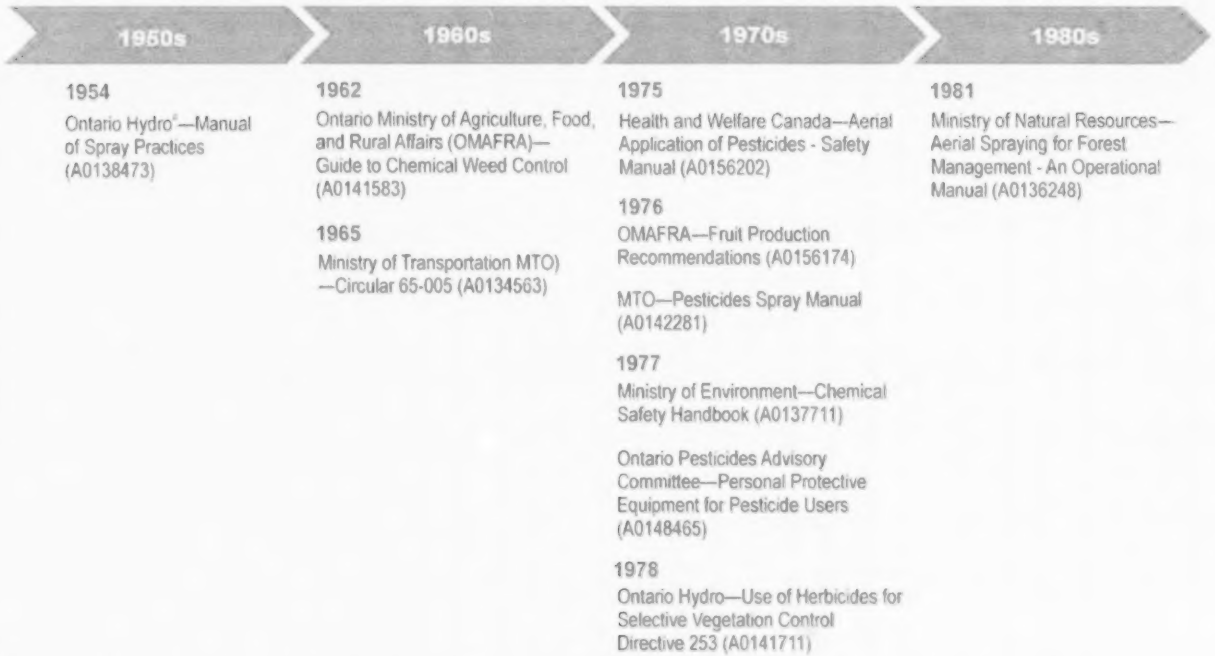
> *(a) The MOE publication "personal Protective Equipment for Pesticides Users" provides information on the selection, suppliers and use of equipment.*
>
> *Masks and goggles (or full face respirators), neoprene gloves, safety toe boots, rainsuits or waterproof coveralls should be used by all personnel in direct contact with the pesticide, even with microbial sprays. Disposable coveralls, now commercially available at low cost, are recommended for project personnel, especially those working at the mixing and loading site....Staff located in the spray blocks should also be equipped with a respirator, goggles and disposable coveralls to prevent skin contact with spray.*
>
> *(b) Fire extinguishers and first aid kits should be available at the mixing site.*
>
> *(c) A large supply of clean water (minimum 200 L) and soap must be located at the mixing site to wash up in the event of accidental chemical spill.*
>
> *(d) Ministry approved hardhats with chinstraps should be worn at all times by all operational personnel* (A0136248, Page 10).

The aerial spray manual also included the following safety equipment checklists:

> *For mixing crew*
>
> *Neoprene safety boots*
> *Neoprene gloves, elbow length*
> *Full rain suits and disposable coveralls*
> *Hard hats with chin straps*
> *Respirators, full face or half face*
> *Safety goggles*
> *Pesticide cartridges with particulate filters*
> *Refills for respirators*
> *First aid kit*
>
> *For balloon men*
>
> *Disposable coveralls*
> *Hard hats with chin straps*
> *Goggles*
> *Respirators, half face*
> *Pesticide cartridges with particulate filters*
> *Safety boots* (A0136248, Page 47).

Figure 5.4 summarizes the development of standard operating procedures for preparing, loading, mixing, applying, storing, transporting, and disposing of pesticides.

Figure 5.4. *Key dates for the development of standard operating procedures for preparing, loading, mixing, applying, storing, transporting, and disposing of pesticides in Ontario.*


1950s
1954
Ontario Hydro*—Manual of Spray Practices (A0138473)
1960s
1962
Ontario Ministry of Agriculture, Food, and Rural Affairs (OMAFRA)—Guide to Chemical Weed Control (A0141583)
1965
Ministry of Transportation MTO) —Circular 65-005 (A0134563)
1970s
1975
Health and Welfare Canada—Aerial Application of Pesticides - Safety Manual (A0156202)
1976
OMAFRA—Fruit Production Recommendations (A0156174)
MTO—Pesticides Spray Manual (A0142281)
1977
Ministry of Environment—Chemical Safety Handbook (A0137711)
Ontario Pesticides Advisory Committee—Personal Protective Equipment for Pesticide Users (A0148465)
1978
Ontario Hydro—Use of Herbicides for Selective Vegetation Control Directive 253 (A0141711)
1980s
1981
Ministry of Natural Resources—Aerial Spraying for Forest Management - An Operational Manual (A0136248)

**The names of organizations referred to in this report have changed several times during the more than three decades of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) use in Ontario. The panel chose to refer to them by their most common or current designation.*

5.4. Standard operating procedures for storing, transporting, and disposing of herbicides

Ontario Hydro's 1954 Manual of Spray Practices said dumping excess herbicides was acceptable as long as it did not contaminate water supplies, livestock feed, trees, shrubs, flowers, or crops. The same standard held for disposing of empty containers (A0138473, Page 71).

By 1962, Ontario Hydro had refined direction for disposing of empty containers: "The utmost care must be taken in the disposal of empty spray containers. Besides the danger to children and animals by contacting these chemicals, a bad impression is created on the public by littering the countryside" (A0141556, Page 18).

The Report of the Minister of Agriculture for the year ending March 31, 1964 advised:

> *...if the container is empty, it should be completely destroyed or better still buried, so there is no possibility of its being reused for some other purpose. If some material is left in the can, it should be stored in a safe place - well out of the reach of children. Materials should never be stored in other than the original container as it is all too easy to forget what is in an unmarked can or bottle. Containers should be tightly closed and kept in a location not readily available to irresponsible persons. The storage should be well away from feed or fertilizer materials to prevent the possibility of contamination by spillage or even vapours from containers. If possible, keep the storage locked* (A0175800, pages 1-2).

In 1972, the presiding committee chair submitted the Report by the Committee Appointed to Study Classification, Transportation, Distribution, Storage and Sale of Pesticides in Ontario to the Minister of Environment (A0172631). Most of this report's storage and transport recommendations referred to vendors as opposed to users.

Category B pesticides (including 2,4,5-T) were only to be sold or transferred to:

> *(a) another premise with a Vendor License Class I or a Vendor Class License II*
>
> *(b) a duly licensed exterminator*
>
> *(c) persons who have obtained a permit for purchase from the Ontario Department of Environment or its designates*
>
> *(d) agriculturists*
>
> *(e) a holder of a bulk license ("Note: exceptions to government and research institutions are recognized")*
>
> *The sale or transfer of Category B pesticides to end users from licensed premises shall be recorded at that time of transfer in a form consistent to that described hereafter as "Vendor Transfer Records"* (A0172631, pages 15-16).

In 1975, Health and Welfare Canada published Aerial Application of Pesticides—Safety Manual, which included the following rules for chemical safety:

> 1. *Inspect pesticide containers for leaks before handling them.*
> 2. *Do not handle containers roughly or carelessly.*
> 3. *Should a leak or spill occur, keep people and animals away from the area; report immediately and decontaminate thoroughly.*
> 4. *Inspect vehicles for contamination after unloading; do not permit a contaminated vehicle to leave until it has been decontaminated.*

5. *Do not store pesticides or empty pesticide containers anywhere near food or drink (including that for animals).*
6. *Do not keep food, drink, tobacco, cups or cutlery anywhere in work area or in work clothes.*
7. *Do not eat, drink, or smoke in work area.*
8. *Do not rub the eyes or touch the mouth while working.*
9. *Wash hands before eating, drinking, smoking, or using the toilet.*
10. *Wear clean rubber gloves and recommended protective clothing when handling pesticides, and an approved respirator where recommended.*
11. *Never fail to dispose of contaminated clothing or faulty protective covering, especially gloves.*
12. *Read the label carefully; if seeking medical aid, take label for the physician's information.*
13. *Have wash facilities as close as possible to the mixing and to the loading sites* (A0156202, Page 9).

The 1976 OMAFRA Fruit Production Recommendations included a safe use of pesticides list:

7. *Store pesticides away from children, irresponsible persons, pets and livestock. Destroy containers. Burn containers that can be burned, and stay out of the smoke. Wash out glass and metal containers, break them and bury them in the municipal dump. Consult the label.*
8. *Excess pesticide solution should be poured out in an isolated area where it will not contaminate crops or water, or injure domestic animals or wildlife* (A0156174, Page 5).

The 1976 MTO Pesticides Spray Manual listed the following storage safety practices (but did not describe how to dispose of containers):

(i) *Store pesticides in their original containers and never use pesticide containers for storing other materials.*

(ii) *Store and handle all containers so that labels remain intact and legible.*

(iii) *Keep containers out of reach of unauthorized persons, especially children.*

(iv) *Never store spray materials near fertilizers, grass seed or where there is a chance of contaminating water supplies.*

(v) *Dispose of all containers when empty* (A0142281, Page 7).

The 1977 and 1980 MOE chemical safety handbooks (A0137711, A0138177) advised the following:

Storage

1. *Pesticides should always be stored in a cool, dry, locked well-ventilated area without floor drains.*
2. *Store away from food and drink used for humans and animal consumption.*
3. *Herbicides should be stored separately from other types of pesticides to prevent cross-contamination.*
4. *Always store pesticides in their original containers.*
5. *A sign with the words "Chemical Storage Warning – Authorized Persons only" in block letters clearly visible should be placed on the outside of each entrance leading into the storage area. Protective clothing, a respirator, and a first aid kit should be available in the storage area.*

Disposal

All empty containers and unused amounts of pesticide must be securely held in an isolated storage area until they can be disposed of correctly. Immediately after emptying a container, rinse several times with the same diluent as used for mixing. Pour the rinse into the spray tank load. Crush empty pesticide containers and bury under at least 18 inches of soil and away from water (A0137711, Page 39; A0138177, Page 46).

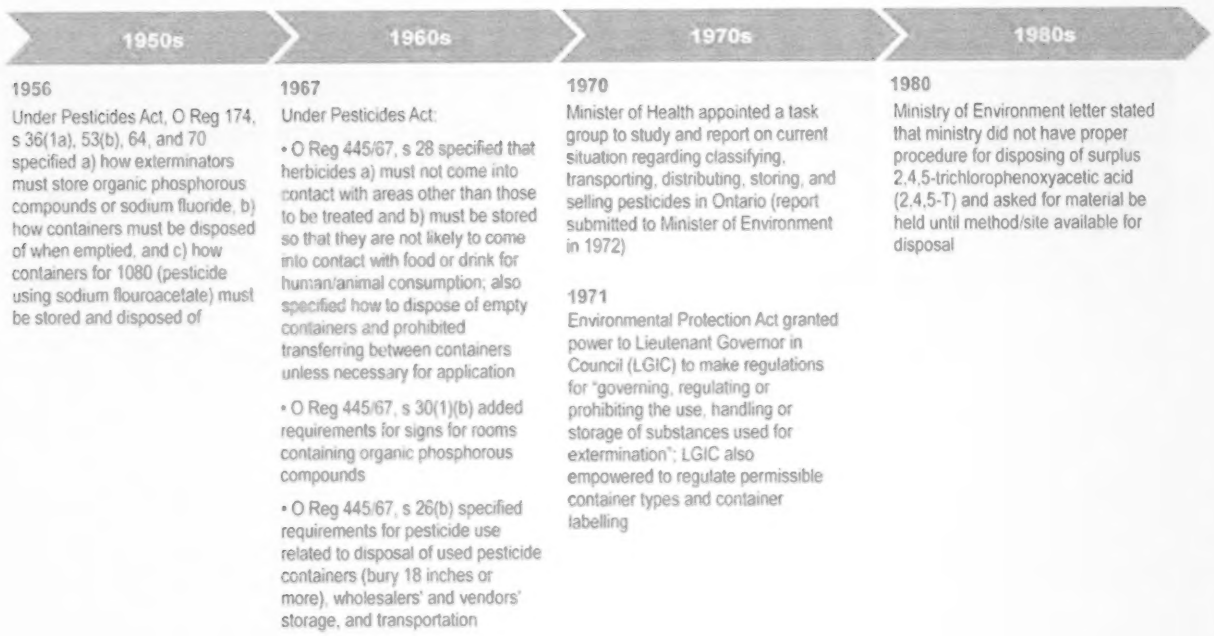
The above storage and disposal information was repeated in Class 1 Land Exterminators Study Guide Draft 1 March 1978 (A0137712, Page 58).

Although the 1981 MNR Aerial Spray Manual was finalized after 2,4,5-T was banned in Ontario, much of its contents was a compilation of practices that developed over the previous three decades of use. These recommendations for storage are summarized below:

- must store chemicals in a separate area that is ventilated and has clear warning signs
- must have sufficient means to clean up spills on storage sites
- must have protective gear available on site (A0131172, Page 40)

Figure 5.5 presents key regulatory dates for storing, disposing of, and transporting herbicides in Ontario.

Figure 5.5. *Key legislative regulatory dates for storing, disposing of, and transporting herbicides in Ontario.*


1950s
1960s
1970s
1980s
1956
Under Pesticides Act, O Reg 174, s 36(1a), 53(b), 64, and 70 specified a) how exterminators must store organic phosphorous compounds or sodium fluoride, b) how containers must be disposed of when emptied, and c) how containers for 1080 (pesticide using sodium flouroacetate) must be stored and disposed of
1967
Under Pesticides Act:
• O Reg 445/67, s 28 specified that herbicides a) must not come into contact with areas other than those to be treated and b) must be stored so that they are not likely to come into contact with food or drink for human/animal consumption; also specified how to dispose of empty containers and prohibited transferring between containers unless necessary for application
• O Reg 445/67, s 30(1)(b) added requirements for signs for rooms containing organic phosphorous compounds
• O Reg 445/67, s 26(b) specified requirements for pesticide use related to disposal of used pesticide containers (bury 18 inches or more), wholesalers' and vendors' storage, and transportation
1970
Minister of Health appointed a task group to study and report on current situation regarding classifying, transporting, distributing, storing, and selling pesticides in Ontario (report submitted to Minister of Environment in 1972)
1971
Environmental Protection Act granted power to Lieutenant Governor in Council (LGIC) to make regulations for "governing, regulating or prohibiting the use, handling or storage of substances used for extermination"; LGIC also empowered to regulate permissible container types and container labelling
1980
Ministry of Environment letter stated that ministry did not have proper procedure for disposing of surplus 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and asked for material be held until method/site available for disposal

5.5. Formal training and education programs

In March 1949, the first course in chemical spraying was given at the Ontario Hydro training centre (A0141556, Page 14).

During the early to mid-1960s, MTO had a training/examination program that allowed employees to qualify to be a landscape crewman and highway inspection assistant (A0175658, Page 11).

In 1967, MOH developed a correspondence course for government employees. Previously, government employees were exempt from license requirements. Those who completed the course in time for the 1968 spray season were issued a temporary license (A0134709, Page 2). The Pesticide Advisory Board developed standards and an exam and granted a license to those who did well on the course/exam and a temporary one-year license to those with lower passing marks (A0136719, A0136690). In 1973, no exams were conducted, but licenses were issued anyway (A0141426).

In Lesson 3, the course covered protecting the applicator as summarized below:

- use the minimum product required
- avoid fumes; mix in open air
- avoid overexposure to spray
- wear protective clothing, including rubber gloves and goggles
- wash skin that has been exposed
- wash clothing often, preferably everyday
- change clothes if spilled on
- do not smoke or eat during application
- wash after application and before smoking or eating (A0135137, Page 7)

In 1967, Ontario Hydro advised that "2,4-D and 2,4,5-T are both harmless to man and animals" (A0138489, Page 9) and that "All spray operations are supervised by expertly trained men. Before a Forestry tradesman engages in this type of work, he must attend a 3 week course in Chemical spraying" (A0138489, Page 13).

A March 1968 Ontario Hydro memo to all area managers advised that "in order to comply with the contents of the Act (Pesticides Act 1967) sufficient personnel in each Area must apply for, attend a short course, write the examination and be in possession of the appropriate license as described within Ontario Regulation 445/67 during all Chemical Brush Control…operations" (A0175657, Page 1).

The course and exams were two days long for 17 people at a time. Exams were held the afternoon of the second day (A0175657, Page 1). Individuals applying for an exterminator's license were to be examined by "a duly qualified medical practitioner." Two letters of reference were to accompany each application for an exterminator's license (A0175657, Page 7).

During the early to mid-1970s, MOE developed a correspondence course entitled Herbicides - General Aspects of Chemical Vegetation Control. Under Precautions, applicators were advised to read and understand the precautions on the label:

> *To protect the applicator:*
>
> *a) Use the minimum rate of the least toxic material that will do the intended job;*
>
> *b) Avoid over-exposure to fumes. Handle or mix volatile materials in the open;*
>
> *c) Avoid over exposure to possible spray drift and wear protective clothing, including rubber gloves and goggles, when handling or applying the more toxic herbicides.*
>
> *Thoroughly wash any splashed materials from exposed areas of the skin. If the herbicides have been splashed into the eyes, immediately wash with large amounts of clean water.*
>
> *Wash clothing frequently, preferably each day after herbicides have been handled or applied. Clothes should be changed immediately if they become contaminated by spilled or splashed material.*
>
> *d) Do not smoke or eat while handling or applying herbicides, and thoroughly wash hands and face before smoking* (A0138195, Page 15; A0138196, Page 15).

The correspondence course also described "protecting other forms of life" by following these directions:

> *a) Store herbicides in original containers. Do not transfer to other containers unless properly labeled, and never use herbicide containers for storing other materials.*
>
> *b) Keep open containers out of the reach of unauthorized persons, children in particular.*
>
> *c) Never store herbicides near fertilizers, crop seeds, or other pesticides.*
>
> *d) Destroy all containers when empty. Paper and cardboard containers may be burned, and metal and glass containers should be mutilated or broken and buried* (A0138195, pages 15-16; A0138196, pages 15-16).

In March 1978, MOE prepared the first draft of the Class 1 Land Exterminator's Study Guide (A0137712, Page 28). It included a safety fact sheet with the following comments about pesticide safety:

> *All pesticides are poisons and while herbicides are perhaps the least toxic group they deserve to be handled with respect. They have been designed to kill plants but can also be harmful to man and other organisms. For this reason, pesticides must always be treated with caution, and all persons having contact with them must be well versed in pesticide safety* (A0137712, Page 28).

This fact sheet appears to have been included in the Class 1 Land Exterminator's Study Guide and repeated the safety information outlined in the 1977 MOE Chemical Safety Handbook (A0137711). The fact sheet also says:

> *19. Coveralls should be worn on the outside of gloves and boots and be long enough to cover wrist. This prevents pesticide from running along the material and into boots or gloves.*
>
> *20. Hats, boots, gloves should be washed daily and hung up to dry.*
>
> *21. Hats, boots, gloves should not be fabric lined as they are hard to clean.*
>
> *22. Always store pesticides in original labeled container, not in empty food containers etc.*
>
> *23. Use care in mixing pesticides. Be sure they are compatible* (A0137712, pages 34-35).

The March 1978 Class 1 Land Exterminator's Study Guide Draft 1 included the following information about labelling under the Pest Control Products Act:

> *Each pesticide must bear the registration number as part of its accepted label. The label must include the brand and name of the chemical, the product's chemical composition and form, the net contents, precautionary measures, and guarantee.*
>
> *The label for each pesticide on the market must bear precautionary statements relative to toxicity. For very toxic poisons, the label includes the handling precautions, diagnostic symptoms of poisoning, and first-aid measures. It should be carefully studied before the use of the product* (A0137712, Page 5).

The draft study guide also provided the following interpretation of O Reg 618/74 (under the Pesticides Act of 1973):

> a) *"adequate respiratory protection" means a respiratory device or devices which effectively protects the user from adverse effects which might result from breathing in of a pesticide during the handling or use of the pesticide;*
>
> b) *"adequate protective clothing" means clothing including rubber or neoprene boots, gloves, hats, coats and other garments that effectively protect the user from adverse effects which might result from a pesticide coming in contact with the skin during the handling or use of the pesticide* (A0137712, Page 16).

Finally, the draft study guide provided guidelines for a range of duties related to pesticides including:

Supervision of equipment:

> *An exterminator shall not supervise an extermination or exterminations for which a total of more than four pieces of pesticide equipment are being used at any time. Whenever an extermination... is being performed and the exterminator is not present, he shall ensure that a person at least sixteen years of age carrying a certificate signed by the exterminator certifying that the person is competent to perform the extermination is present and in charge of each piece of pesticide application equipment* (A0137712, Page 22).

Storage and disposal of containers:

> *No person other than a wholesale vendor or a limited wholesale vendor shall have in his possession a pesticide other than in the container in which it was originally offered for sale* (A0137712, Page 19).
>
> *An empty container that has been used to hold a Schedule 1, 2, or 5 pesticide shall be disposed of by puncturing or breaking and burying the container in such a manner that it is covered by at least eighteen inches of soil and is not near any watercourses or water table* (A0137712, Page 20).

Handling broken containers:

> *Where the original container of Schedule 1, 2, or 5 pesticide is damaged or broken, the person responsible for the pesticide shall,*
>
> *a) replace the container with a container equivalent to that originally used; or*
>
> *b) dispose of the container and its contents by burying them under eighteen inches of soil in such a manner that they are not near any watercourse or water table; and*
>
> *c) clean up any spillage and decontaminate any area, carrier or commodity that has come in contact with the pesticide* (A0137712, Page 20).

Preventing contact with food and drink:

> *No person shall store any pesticide in such a manner that the pesticide is likely to come into contact with food or drink intended for human or animal consumption* (A0137712, Page 23).

Storing

> *Every person responsible for a Schedule 1,2, or 5 pesticide will ensure that:*
>
> *(a) any room in which the pesticide is stored is ventilated to the outside atmosphere;*
>
> *(b) a placard is affixed and maintained on the outside of each door leading into the room in which the pesticide is stored bearing the words "Chemical Storage Warning – Authorized Persons Only" in block letters clearly visible; and*
>
> *(c) no person can enter the room in which the pesticide is stored without the express permission of the person responsible* (A0137712, Page 23).

Everyone should store pesticides:

> *(a) in such a manner that the pesticide will not likely contaminate food or drink intended for human or animal consumption;*
>
> *(b) in such a manner that the pesticide will not be likely to impair the health or safety of any person;*
>
> *(d) in an area near which there is prominently displayed a list of emergency telephone numbers, including those of the local fire department, hospital and poison control centre; and*
>
> *(e) in an area that is maintained in a clean and orderly manner; and*
>
> *(c) in an area near which adequate respiratory protection and adequate protective clothing are kept readily available by the licensee for emergency purposes; and*
>
> *(d) That has no floor drain that leads into or drains directly or indirectly into a storm sewer, sanitary sewer or watercourse* (A0137712, pages 23-24).

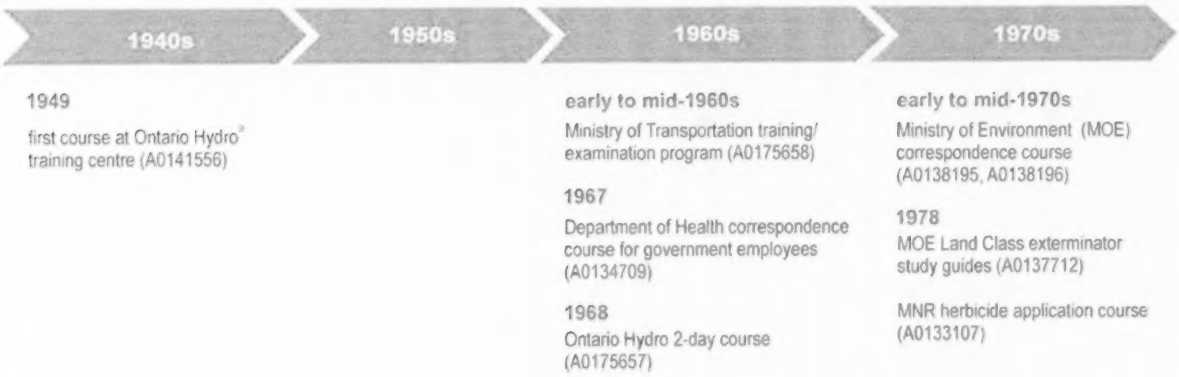
Transporting pesticides:

> *No operator shall permit a vehicle to be used in transporting or applying a pesticide to be used in connection with a land extermination performed by a person licensed to perform land exterminations...unless a plastic identification sticker is obtained from the Director and is affixed to the rear of the vehicle in such a manner that the plate is visible and legible at all times* (A0137712, Page 22).

By 1978, MNR appeared to have developed its own herbicide application course. A 1978 MNR memo to district managers said, "A short herbicide application course (such as was offered by Pest Control in Kapuskasing in July) should be slated for Cochrane, or alternatively, Pest Control staff should visit this district during next year's program. Similarly a visit to Thunder Bay District may prove beneficial in terms of improving Ministry aerial application programs" (A0133107, Page 17).

Figure 5.6 presents a timeline for when various provincial organizations developed formal training and education programs.

Figure 5.6. *Timeline for development of formal provincial training and education programs for applying herbicides in Ontario.*



[a] *The names of organizations referred to in this report have changed several times during the more than three decades of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) use in Ontario. The panel chose to refer to them by their most common or current designation.*

6. Risk assessment

All substances can be toxic at some level of exposure, but the amount, frequency, and duration of exposure that result in toxicity vary from substance to substance. Thus, the likelihood a substance will pose a risk under typical conditions of use or occurrence is a function of both toxicity and exposure. Risk assessment describes how toxic a chemical is, what exposure results from its various uses, what the probability of harm from use is, and how to characterize that risk.

All substances can be toxic at some level of exposure. Risk assessment describes how toxic a chemical is, what exposure results from its various uses, what the probability of harm from use is, and how to characterize that risk.

Assessing risk is fraught with considerable uncertainty, and applying this imperfect process to decisionmaking can be difficult. However, structured risk assessment provides a transparent and disciplined approach to informing the decisionmaking process for the regulation of potentially toxic substances.

As described in Chapter 2, risk assessment involves 4 steps (NAS 2009):

- identifying hazard
- assessing dose response
- assessing exposure
- characterizing risk

For the assessment of the use of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) in Ontario, including its contaminant 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), the panel adopted the U.S. National Academy of Sciences' risk assessment paradigm (NAS 2009; Figure 6.1). Risk assessment is often followed by risk *management* (as noted in Figure 6.1), but the latter is based on not just scientific considerations but also social, economic, and legal issues as part of regulatory and policy decisionmaking (California Department of Pesticide Regulations 2012). For the 2,4,5-T and TCDD assessment, the panel did not consider risk management, as it was outside its mandate.

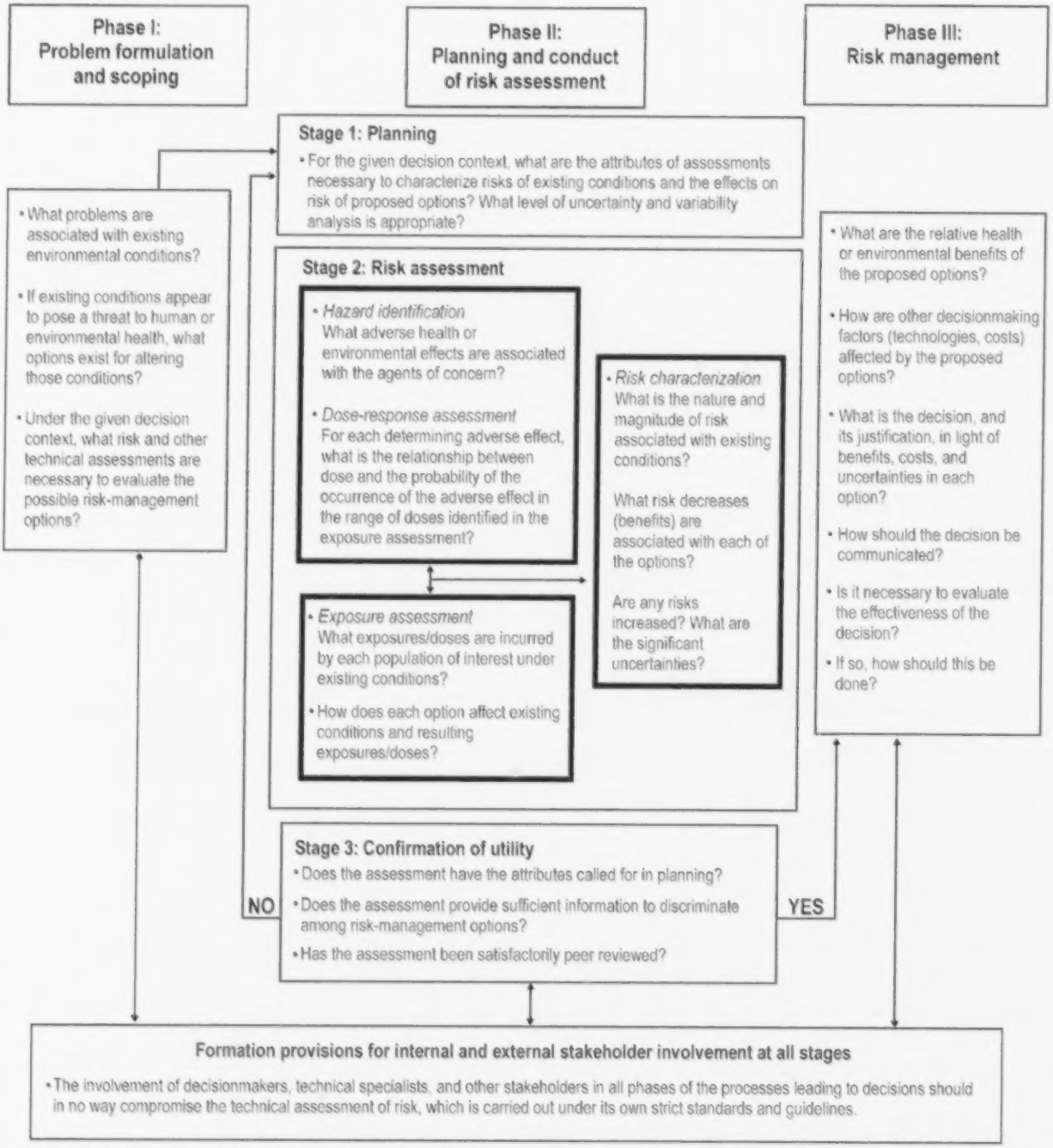

Phase I:
Problem formulation and scoping
Phase II:
Planning and conduct of risk assessment
Phase III:
Risk management
Stage 1: Planning
• For the given decision context, what are the attributes of assessments necessary to characterize risks of existing conditions and the effects on risk of proposed options? What level of uncertainty and variability analysis is appropriate?
• What problems are associated with existing environmental conditions?
• If existing conditions appear to pose a threat to human or environmental health, what options exist for altering those conditions?
• Under the given decision context, what risk and other technical assessments are necessary to evaluate the possible risk-management options?
Stage 2: Risk assessment
• Hazard identification
What adverse health or environmental effects are associated with the agents of concern?
• Dose-response assessment
For each determining adverse effect, what is the relationship between dose and the probability of the occurrence of the adverse effect in the range of doses identified in the exposure assessment?
• Exposure assessment
What exposures/doses are incurred by each population of interest under existing conditions?
• How does each option affect existing conditions and resulting exposures/doses?
• Risk characterization
What is the nature and magnitude of risk associated with existing conditions?
What risk decreases (benefits) are associated with each of the options?
Are any risks increased? What are the significant uncertainties?
• What are the relative health or environmental benefits of the proposed options?
• How are other decisionmaking factors (technologies, costs) affected by the proposed options?
• What is the decision, and its justification, in light of benefits, costs, and uncertainties in each option?
• How should the decision be communicated?
• Is it necessary to evaluate the effectiveness of the decision?
• If so, how should this be done?
Stage 3: Confirmation of utility
• Does the assessment have the attributes called for in planning?
• Does the assessment provide sufficient information to discriminate among risk-management options?
• Has the assessment been satisfactorily peer reviewed?
NO
YES
Formation provisions for internal and external stakeholder involvement at all stages
• The involvement of decisionmakers, technical specialists, and other stakeholders in all phases of the processes leading to decisions should in no way compromise the technical assessment of risk, which is carried out under its own strict standards and guidelines.

Figure 6.1. *A framework for risk-based decisionmaking that maximizes the utility of risk assessment (reprinted with permission from NAS 2009).*

6.1. Characterizing risk

To characterize possible risk to herbicide applicators, bystanders, recreational and sustenance land users, and local residents, the panel had to assess the hazard that the chemical could pose (hazard identification), estimate the dose at which adverse effects are observed (dose-response assessment), and then compare this dose with the dose (exposure) to which various population subsets may have been exposed (exposure assessment). Assessment results are often expressed using either a *margin of safety approach* or a *slope factor approach* (described below).

6.1.1. Margin of safety approach to characterizing risk

The margin of safety assessment is typically used when assessing substances that produce a non-linear dose response, particularly when the dose is very low or at the low end of the range of expected human exposure, and that are not known to be genotoxic. Margin of safety is also known as *hazard quotient* (US EPA 2005).

Margin of safety is calculated by dividing the *no observed adverse effect level* (NOAEL), the highest dose at which adverse effects were not observed in a test population (hazard identification), by the estimated human exposure. If the NOAEL is based on a study using experimental animals, the benchmark margin of exposure = 100. A ratio of 100 is typically used to indicate that the effect is reasonably certain not to occur in exposed people, even if exposure occurs every day over an entire lifetime.

This approach can also be applied to a *toxicological reference value* (TRV; estimate of the daily dose of a substance considered safe, even with daily exposure over an entire lifetime) or *tolerable daily intake* (TDI[1]; estimate of amount of substance that can be consumed daily over a lifetime without appreciable health risk), which includes applying an uncertainty factor to the NOAEL. When the expected human exposure is divided by the TRV, a benchmark ratio of no greater than 1 is typically used to indicate that the effect is reasonably certain not to occur in exposed people, even if exposure occurs every day over their lifetime. Even if the ratio is greater than 1, which indicates a potential for adverse effects, they may not occur (US EPA 2005).

6.1.2. Slope factor approach to characterizing risk

The margin of safety approach is not appropriate for substances thought to be genotoxic. The alternative involves deriving the toxicological reference dose (TRD) to estimate the upper bound of the slope between exposure and effect occurrence. The slope of the dose-response relationship (exposure/dose) is known as the *slope factor*. When multiplied by the exposure level (dose or concentration, as appropriate), this factor provides the upper limit of the probability of cancer in a chronically exposed population (Health Canada 2010).

6.2. Determining tolerable intake for the chemicals of concern

The United Nations Joint FAO/WHO Expert Committee on Food Additives concluded that "...a tolerable intake could be established for TCDD on the basis of the assumption that there is a threshold for all effects, including cancer. Carcinogenicity due to TCDD was not linked to mutagenicity or DNA binding, and it occurred at higher body burdens in animals than other toxic effects...." The committee also concluded that "... the establishment of a tolerable intake based on effects other than cancer would also address any carcinogenic risk" (WHO 2002).

The establishment of a tolerable intake for TCDD based on effects other than cancer would also address any carcinogenic risk.

[1] The panel refers to TRV and TDI interchangeably, based on what was used in the source document.

This committee derived a tolerable monthly TCDD intake of 74 pg/kg BW/month, equivalent to a TDI of 2.3 pg/kg BW/day (BW = body weight). Health Canada and the Ministry of the Environment (MOE)[2] adopted the same tolerable intake of 2.3 pg/kg BW/day (Health Canada 2010, MOE 2011). In its recent re-assessment, the U.S. Environmental Protection Agency (U.S. EPA) derived a lower TDI for TCDD—0.7 pg/kg BW/day (US EPA 2012. Given the uncertainty associated with deriving a tolerable intake for TCDD, the panel does not consider there to be a meaningful biologically relevant difference between the TDI established by the United Nations, Health Canada, and MOE (2.3 pg/kg BW/day) and that derived by the U.S EPA (0.7 pg/kg BW/day) . Therefore, the panel chose to adopt the TDI (and underlying biological basis) used by the governments of Canada and Ontario and the Joint FAO/WHO Expert Committee on Food Additives, particularly the latter's view that basing the TDI on effects other than cancer would adequately protect humans from carcinogenic risk as well.

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The panel also established the following TDI amounts for the parent herbicide, 2,4,5-T:

- Acute exposure: 20 µg/kg BW/day for 2,4,5-T (Newton and Norris 1981)
- Chronic exposure: 10 µg/kg BW/day (US EPA 1989)

TRVs are generally used to establish safe exposure levels for a general population and not for occupationally exposed sub-populations who may have highly variable exposure patterns (Health Canada 1994). In all cases, the panel relied on TRVs published by recognized regulatory and international agencies or in the scientific literature. The panel felt that deriving new TDIs was beyond its mandate and was satisfied with the process and basis others used to derive TDIs. Table 6.1 (repeated from Chapter 3) summarizes the TRVs the panel used.

Table 6.1. *Toxicological reference values (tolerable daily intake) by endpoint for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Source	Chemical of interest	Endpoint	Reference value[a]
Newton and Norris 1981	2,4,5-T	acute	20 µg/kg BW/day
US EPA 1989		chronic	10 µg/kg BW/day
US DHHS 1998	TCDD	acute	200 pg/kg BW/day
Health Canada 2010, MOE 2011, WHO 2002		chronic	2.3 pg/kg BW/day
US EPA 2012		chronic	0.7 pg/kg BW/day

[a] *BW = body weight.*

[2] In most cases, the names of organizations referred to in this report have changed several times during the more than three decades covering the period of use of 2,4,5-T in Ontario. The panel chose to refer to them by their most common or current designation.

As noted in Section 6.1.1, exposure assessment is a key component of risk assessment and the panel's undertaking. The use of 2,4,5-T in Ontario spanned approximately 30 years, with all use curtailed by 1979.

The panel adopted a well-documented and widely practiced risk assessment protocol (IOM 2012) to assess the hazards of 2,4,5-T and TCDD, to evaluate the exposure potential for various Ontario populations that could have arisen as a result of use by Ontario government departments and agencies, and to assess the conditions of exposure under which adverse effects have been observed in laboratory animal studies and in human populations. While the panel identified extensive toxicological and epidemiological information on the hazards of the parent herbicide and the TCDD contaminant, and the exposure circumstances under which adverse effects have been observed, they could not locate any exposure measurements among the Ontario population specific to use by Ontario government departments and agencies.

To estimate likely human exposure to 2,4,5-T/TCDD during the period of Ontario government use, the panel thoroughly assessed use records submitted by various departments and agencies. They found that while many Ontario government departments used 2,4,5-T, the Ministry of Natural Resources (MNR), Ministry of Transportation (MTO), and Ontario Hydro were the primary users. The panel noted that municipal users applied significantly more 2,4,5-T than the Ontario government did (Figure 6.2, reproduced from Chapter 2).

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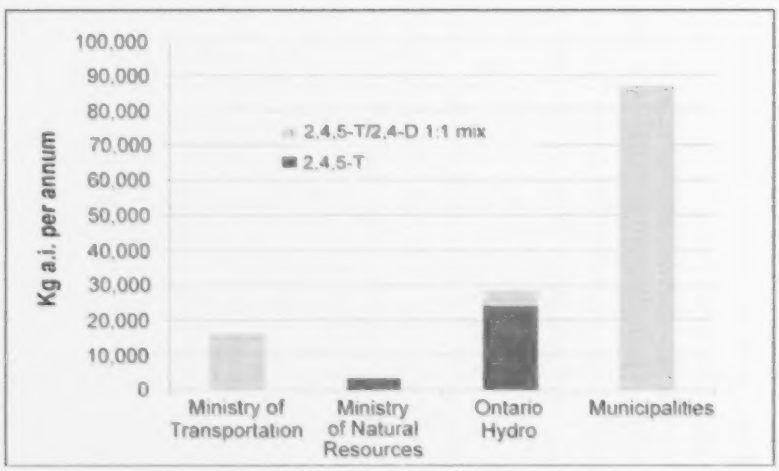

Figure 6.2. *Annual[a] use of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) by provincial government agencies, as reported in the 1978 Ontario Pesticides Advisory Council report Review of the Use of 2,4-D, Other Phenoxy Herbicides and Picloram by Ontario Government Agencies (A0133096).*

[a]The panel was unable to determine what year annual referred to and how representative these use patterns were throughout the period of use of 2,4,5-T in Ontario.

6.3. Exposure estimates

As noted elsewhere in this report, the objective of the exposure assessment was to predict potential human exposure to 2,4,5-T and its contaminant TCDD using available documentation to develop the exposure scenarios and pathways identified below. The panel's estimation of exposure to the chemicals of concern is based on the following parameters:

- use patterns and application rates of the herbicides as provided by government records in the MNR database
- occupational and non-occupational activities as defined by government operation manuals, standards, pesticide handler assumptions, and information on the nature and location of herbicide use in Ontario
- physical/chemical characteristics of the chemicals of concern, which determine the interaction and behaviour of a chemical with its surrounding environment (water solubility, volatility, tendency to bind to particles, etc.)
- characteristics of the environmental compartments affected by spraying (air, soil, plants, etc.), as well as the quantities and persistence of chemicals entering the compartments from various sources
- behavioural characteristics of the human receptors that determine the actual exposures via interaction with the various pathways (e.g., respiration rate, body weight)
- equations and algorithms used to predict receptor exposure

Whenever possible, physiological factors such as body weight and inhalation rate and behavioural factors such as incidental soil ingestion were selected to be consistent with Health Canada and MOE (Health Canada 2010, MOE 2011). Occupational and bystander exposures were estimated based on historical spray information. Historical spray information and exposure point concentrations in various environmental media (soil, dust, wild berries), used for the recreational, residential, and First Nations assessments, were predicted using the equations and assumptions described in Chapter 4 of this report.

The exposure assessment provided a range of potential exposures for each scenario, represented by three individual estimates: low, central, and high. The high estimates were based on a series of worst-case assumptions, applied one after another, introducing a repetitive input parameter selection bias. Similarly, whenever possible, the central and low exposure estimates were based on central (average) and low-end exposure parameters, respectively. Parameters that vary within the exposure estimates include:

- TCDD content of 2,4,5-T
- use of personal protective equipment
- fraction of applied herbicide penetrating the forest canopy
- half-lives of TCDD and 2,4,5-T in soil

Not all input parameters were associated with a range (i.e., low, central, and high). Body weight and soil ingestion, for example, were assigned a single value for each exposure estimate. A range of potential exposure values (determined by selected low, central, and high input parameters) were developed to reflect the uncertainty and variability of the quantitative exposure estimates. For more details on the methods and approach used to estimate human exposure, see Chapter 4 of this report.

As discussed above, the panel adopted a margin of safety approach to characterize the risk to various populations from Ontario government departments' and agencies' use of 2,4,5-T. To describe the margin of safety for 2,4,5-T and TCDD dose levels in various studies that did not result in adverse toxicological effects (TRVs) and various populations' exposure levels, the panel directly compared these variables with each other. To ensure exposure frequency and duration were properly characterized, the panel considered both acute and chronic effects. The human exposure estimates (tables 6.2 to 6.13) were then directly compared with the TRVs (or TDIs) published in the scientific literature (tables 6.14 to 6.25). As noted above, these values were:

- For acute effects:
  - 2,4,5-T: 20 µg/kg BW/day (Newton and Norris 1981)
  - TCDD: 200 pg/kg BW/day (US DHHS 1998)
- For chronic effects:
  - 2,4,5-T: 10 µg/kg BW/day (US EPA 1989)
  - TCDD: 2.3 pg/kg BW/day (WHO 2002, Health Canada 2010, MOE 2011)

The panel concluded that bystander exposures to 2,4,5-T and TCDD were likely short-term or *acute*. All other exposures, including occupational, recreational and residential, were considered *chronic*.

Human exposure estimates are provided in Chapter 4 and reproduced here for convenience:

- Tables 6.2- 6.7: occupational exposure estimates for:
  - 6.2: MTO applicators
  - 6.3: Ontario Hydro—aerial application
  - 6.4: Ontario Hydro—ground application
  - 6.5: MNR—aerial application
  - 6.6: MNR—ground application
  - 6.7: MNR—backpack application
- Tables 6.8-6.11: exposure estimates for:
  - 6.8: recreational users
  - 6.9: residents
  - 6.10: fish consumers
  - 6.11: game consumers
- Tables 6.12-13: exposure estimates for bystanders:
  - 6.12: following Ontario Hydro direct spray on 1 day/year
  - 6.13: following MTO/MNR direct spray on 1 day/year

In all cases, the coloured values in the tables represent exposures that exceeded the TRV (or TDI) for acute or chronic exposure. In the interest of safety, the panel adopted an approach of modelling acute exposures for bystanders only. All other exposures were considered to have been chronic.

6.4. Margin of safety estimates

Tables 6.14 to 6.25 show the margin of safety estimates, derived by comparing estimated exposures in the assessed populations with either acute or chronic TRVs, as discussed above. The populations described via this method include Ontario Hydro, MTO, and MNR workers (occupational exposure) as well as bystanders. The margins of safety are in coloured font when the margin exceeded the benchmark value of 1.

The tables reveal the following:

- The populations whose exposure levels exceeded the benchmark (1) were almost all chronically exposed Ontario government workers who handled 2,4,5-T and who encountered the central and high levels of TCDD.
- In almost all cases, the TCDD margin of safety exceeded the benchmark only with the highest exposure estimates.
- When central exposure estimates for TCDD exceeded the benchmark, it was by only a small amount.
- Ontario Hydro right-of-way applicators were the only population whose lowest estimated chronic exposures marginally exceeded the benchmark margin of 1.
- Bystander exposures, defined by the panel as being acute, were found to marginally exceed the benchmark margin of 1 only for the Ontario Hydro and MNR scenarios and only for the highest TCDD exposure estimates.
- All MTO bystander exposures to TCDD and all Ontario Hydro and MNR central and low bystander exposures to TCDD did not exceed the benchmark margin.
- Similarly, all bystander exposures to 2,4,5-T for all Ontario Hydro, MNR, and MTO scenarios did not exceed the benchmark margin.

The panel also assessed margins of safety for exposures of non-occupational population sub-groups, including toddlers and adults (tables 6.20 and 6.23). In no case did the margins of safety exceed the benchmark of 1.

The assessment revealed that several occupationally exposed populations may have experienced exposures substantially above the margin of safety. However, such exposures were restricted to occupational populations (Ontario Hydro, MNR, and MTO workers) who were chronically exposed. Non-occupational exposure estimates only marginally exceeded the margin of safety (for bystanders). The TRVs used to assess exposure acceptability erred on the side of safety, as regulatory policy for assessing risk requires, but populations with the highest exposure levels will not necessarily experience adverse health effects. The assessment merely indicates that acceptable margins of safety were exceeded, and people's health *could have been* affected.

The assessment revealed that several occupationally exposed populations may have experienced exposures substantially above the margin of safety. However, such exposures were restricted to occupational populations (Ontario Hydro, MNR, and MTO workers) who were chronically exposed.

As noted above, any association between exposure to 2,4,5-T/TCDD and disease—as described in the scientific literature and Chapter 3 of this report—relates to populations. The panel's assessment did not address whether any individual's health problem is associated with or caused by the chemicals in question. The Institute of Medicine (IOM) found that it is impossible to quantify the degree of risk that exposure to TCDD-contaminated herbicides, including 2,4,5-T, poses to Vietnam veterans (IOM 2007). Since 1994, IOM has published biannual

reports that show a lack of robust information about the relationship between Vietnam veterans' exposure to TCDD-contaminated 2,4,5-T and specific health outcomes (IOM 2012). According to the Institute's 2012 update, the U.S. Congress requires that when no specific exposure information is available, Vietnam veterans should be assumed to have been exposed (IOM 2012). The panel carefully considered the issue of exposure and decided that they could re-create a possible exposure profile for provincial workers and residents with enough certainty to comment on exposure response, a basic premise of toxicological risk assessment.

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The goal of quantitative exposure assessment is to produce a conservative model to ensure that exposure is never underestimated, while recognizing that overestimates may occur. Given the tendency for the assumptions described in this report to result in overestimates, the results likely err on the side of conservatism (safety).

The panel characterized three levels of exposure, driven mainly by TCDD contaminant levels and the nature and degree to which the workers used personal protective equipment. Notably, the panel assumed that TCDD contaminant levels in 2,4,5-T used between 1947 and 1970 in Ontario were equal to the highest reported in the Vietnam-era production batches of 2,4,5-T. These values were 0.05, 13, and 47 ppm, for the low, central, and high estimates, respectively. The panel assumed TCDD contaminant levels after 1970 were consistent with Agriculture Canada directives (A0137447, Page 1; A0137444, Page 1; A0137443, Page 1).

The panel was not able to verify how closely government workers followed guidelines for personal protective equipment. Therefore, they assumed that the low, central, and high estimates of exposure corresponded to the best (low exposure) and worst (high exposure) levels of protection that personal protective equipment could have provided, with the central estimate representing the most realistic case. While Ontario Hydro application rates were the highest of the three government scenarios, even those were up to ten times less than those described for Vietnam (AO138404). The panel considered the central exposure estimate to best represent actual exposures.

Given the results of the exposure assessment and margin of safety comparisons, the panel concluded that a relatively small number of workers in the following categories potentially experienced elevated exposure levels:

- Ontario Hydro and MTO workers involved in ground mixing/loading and application
- MNR workers and junior rangers involved in backpack mixing/loading and application
- MNR workers involved in aerial mixing/loading and flagging

The panel noted that while some bystander exposures exceeded the benchmark margin of 1, the highest estimated exposures corresponded to margins of safety of less than 2. The panel generally considered the acceptable benchmark margin to be 1 or less. However, given the inherent assumptions on which the panel

assessment is based, in those cases where the margin is greater than the benchmark of 1, adverse effects will not necessarily occur. Given the assumptions and uncertainties in the bystander estimates, the acute nature of bystander exposures, and the safety factors inherent in the TRVs used in the evaluation, the panel felt that the margins of safety in the bystander exposure estimates made acute health outcomes very unlikely. The short-term effects associated with these types of short-term exposures are more likely to be transient and reversible than long-term exposures. Given the magnitude of exceedances for bystanders were small, they were not considered biologically relevant; thus, no short-term effects would be expected.

The panel noted that the biological relevance of exposures that exceeded the benchmark margins must be viewed in the proper context. To assess potential risks, the panel found it necessary to re-create and model various exposure scenarios, and re-creating exposure events that occurred decades ago is rife with unknowns and uncertainties. Many assumptions involving large amounts of data and numerical variables were used to derive exposure estimates. Some of these input variables were obtained from the scientific literature, while others were scenario-specific and obtained from various sources of information available from various ministries for the study. Each decision and variable had some degree of uncertainty that could have affected the assessment outcome. The U.S. EPA has noted that even when the margin of safety is greater than the benchmark of 1, adverse effects may not occur (US EPA 2005).

For a summary of the assumptions and uncertainties related to the exposure assessment, see Chapter 4, Table 4.47. This table also presents the potential effects of these assumptions and the uncertainties in the assessment conclusions. When possible, the panel erred on the side of caution, likely resulting in an overestimate of exposure.

In conclusion, the panel's approach was a population level assessment and not intended to describe risks to any specific individual. A population level assessment can neither confirm nor refute the association between an individual's health problem and 2,4,5-T or TCDD exposure, but it provides an indication of the average risk for the population. Individuals will vary widely in their responses to exposures and in the exposures themselves and may have risks above or below the population estimate. Having said this, the panel noted that the IOM has developed a list of diseases for which exposed Vietnam veterans may be at increased risk. The panel agreed that the IOM findings reasonably reflect the current state of epidemiology and toxicology but noted that only a few exposure scenarios in Ontario placed a small potentially exposed population into a risk category. Thus, the associated risk of individuals in these categories developing these diseases as a result of 2,4,5-T or TCDD exposure would likely be very low. And again, an adverse effect will not necessarily occur, even in those cases where the estimated margins exceeded the benchmark.

Table 6.2. *Estimates of Ministry of Transportation occupational exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Year	PHED[a] data—mixer/loader total (µg/kg/day)						PHED data—right-of-way applicator total (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[b]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1953	4.04E-02	5.09E-02	2.77E-01	5.35E-09	1.78E-06	3.67E-05[c]	4.35E-01	5.60E-01	1.69E+00	5.48E-08	1.89E-05	2.19E-04
1954	4.05E-02	5.11E-02	2.78E-01	5.37E-09	1.79E-06	3.68E-05	4.37E-01	5.62E-01	1.69E+00	5.50E-08	1.90E-05	2.20E-04
1955	4.15E-02	5.23E-02	2.84E-01	5.49E-09	1.83E-06	3.77E-05	4.47E-01	5.75E-01	1.73E+00	5.63E-08	1.94E-05	2.25E-04
1956	4.11E-02	5.19E-02	2.82E-01	5.45E-09	1.82E-06	3.74E-05	4.44E-01	5.71E-01	1.72E+00	5.59E-08	1.93E-05	2.23E-04
1957	4.26E-02	5.37E-02	2.92E-01	5.64E-09	1.88E-06	3.87E-05	4.59E-01	5.91E-01	1.78E+00	5.79E-08	1.99E-05	2.31E-04
1958	3.12E-02	3.93E-02	2.14E-01	4.14E-09	1.38E-06	2.84E-05	3.36E-01	4.33E-01	1.30E+00	4.24E-08	1.46E-05	1.69E-04
1959	3.42E-02	4.31E-02	2.35E-01	4.54E-09	1.51E-06	3.11E-05	3.69E-01	4.75E-01	1.43E+00	4.65E-08	1.60E-05	1.86E-04
1960	3.82E-02	4.82E-02	2.62E-01	5.06E-09	1.68E-06	3.47E-05	4.12E-01	5.30E-01	1.60E+00	5.19E-08	1.79E-05	2.07E-04
1961	3.95E-02	4.98E-02	2.70E-01	5.23E-09	1.74E-06	3.59E-05	4.25E-01	5.48E-01	1.65E+00	5.36E-08	1.85E-05	2.14E-04
1962	4.51E-02	5.69E-02	3.09E-01	5.98E-09	1.99E-06	4.10E-05	4.86E-01	6.26E-01	1.88E+00	6.13E-08	2.11E-05	2.45E-04
1963	5.89E-02	7.43E-02	4.04E-01	7.81E-09	2.60E-06	5.35E-05	6.35E-01	8.18E-01	2.46E+00	8.01E-08	2.76E-05	3.20E-04
1964	6.29E-02	7.93E-02	4.31E-01	8.33E-09	2.77E-06	5.71E-05	6.78E-01	8.73E-01	2.63E+00	8.54E-08	2.94E-05	3.41E-04
1965	5.58E-02	7.03E-02	3.82E-01	7.39E-09	2.46E-06	5.07E-05	6.01E-01	7.74E-01	2.33E+00	7.58E-08	2.61E-05	3.03E-04
1966	6.33E-02	7.98E-02	4.34E-01	8.39E-09	2.79E-06	5.75E-05	6.82E-01	8.79E-01	2.65E+00	8.60E-08	2.96E-05	3.44E-04
1967	8.07E-02	1.02E-01	5.53E-01	1.07E-08	3.56E-06	7.33E-05	8.70E-01	1.12E+00	3.37E+00	1.10E-07	3.78E-05	4.38E-04
1968	7.82E-02	9.85E-02	5.36E-01	1.04E-08	3.45E-06	7.10E-05	8.42E-01	1.08E+00	3.27E+00	1.06E-07	3.66E-05	4.24E-04
1969	7.81E-02	9.85E-02	5.35E-01	1.04E-08	3.45E-06	7.10E-05	8.42E-01	1.08E+00	3.26E+00	1.06E-07	3.66E-05	4.24E-04
1970	8.40E-02	1.06E-01	5.76E-01	1.11E-08	3.71E-06	7.63E-05	9.06E-01	1.17E+00	3.51E+00	1.14E-07	3.93E-05	4.56E-04
1971	7.15E-02	9.01E-02	4.90E-01	9.47E-08	1.21E-07	6.91E-07	7.71E-01	9.92E-01	2.99E+00	9.71E-07	1.29E-06	4.13E-06
1972	7.98E-02	1.01E-01	5.47E-01	1.06E-07	1.35E-07	7.71E-07	8.60E-01	1.11E+00	3.33E+00	1.08E-06	1.44E-06	4.61E-06
1973	7.98E-02	1.01E-01	5.47E-01	1.06E-07	1.35E-07	7.71E-07	8.60E-01	1.11E+00	3.33E+00	1.08E-06	1.44E-06	4.61E-06

[a] *PHED = Pesticide Handler Exposure Database.*

[b] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[c] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 6.3. *Estimates of Ontario Hydro occupational exposure (aerial) to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Year	PHED[a] data—applicator—aerial, fixed wing (µg/kg/day)						PHED data—flagman—aerial, fixed wing (µg/kg/day)						PHED data—mixer/loader—aerial, fixed wing (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[b]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1953	6.25E-03	6.25E-03	6.25E-03	6.58E-11	1.71E-08	6.18E-08	1.66E-02	1.81E-02	1.70E-02	1.54E-10	4.44E-08	1.49E-07	3.34E-02	4.22E-02	2.29E-01	3.71E-10	1.23E-07	2.54E-06
1954	1.53E-02	1.53E-02	1.53E-02	1.60E-10	4.16E-08	1.50E-07	4.06E-02	4.41E-02	4.16E-02	3.74E-10	1.08E-07	3.63E-07	8.17E-02	1.03E-01	5.60E-01	9.02E-10	3.00E-07	6.18E-06
1955	2.95E-03	2.95E-03	2.95E-03	3.10E-11	8.06E-09	2.92E-08	7.84E-03	8.52E-03	8.04E-03	7.24E-11	2.09E-08	7.03E-08	1.58E-02	1.99E-02	1.08E-01	1.75E-10	5.82E-08	1.20E-06
1956	9.10E-03	9.10E-03	9.10E-03	9.55E-11	2.48E-08	8.98E-08	2.42E-02	2.63E-02	2.48E-02	2.23E-10	6.45E-08	2.16E-07	4.88E-02	6.15E-02	3.34E-01	5.38E-10	1.79E-07	3.69E-06
1957	5.03E-02	5.03E-02	5.03E-02	5.27E-10	1.37E-07	4.96E-07	1.34E-01	1.45E-01	1.37E-01	1.23E-09	3.56E-07	1.19E-06	2.69E-01	3.39E-01	1.84E+00	2.97E-09	9.89E-07	2.04E-05
1958	5.69E-02	5.69E-02	5.69E-02	5.98E-10	1.55E-07	5.62E-07	1.52E-01	1.65E-01	1.55E-01	1.40E-09	4.04E-07	1.35E-06	3.05E-01	3.84E-01	2.09E+00	3.37E-09	1.12E-06	2.31E-05
1959	4.83E-02	4.83E-02	4.83E-02	5.06E-10	1.32E-07	4.76E-07	1.29E-01	1.40E-01	1.32E-01	1.18E-09	3.42E-07	1.15E-06	2.59E-01	3.26E-01	1.77E+00	2.85E-09	9.49E-07	1.96E-05
1960	6.18E-02	6.18E-02	6.18E-02	6.49E-10	1.69E-07	6.10E-07	1.65E-01	1.79E-01	1.69E-01	1.52E-09	4.38E-07	1.47E-06	3.31E-01	4.17E-01	2.27E+00	3.66E-09	1.22E-06	2.51E-05
1961	5.07E-02	5.07E-02	5.07E-02	5.32E-10	1.38E-07	5.00E-07	1.35E-01	1.47E-01	1.38E-01	1.24E-09	3.59E-07	1.21E-06	2.72E-01	3.42E-01	1.86E+00	3.00E-09	9.98E-07	2.06E-05
1962	4.11E-02	4.11E-02	4.11E-02	4.32E-10	1.12E-07	4.06E-07	1.10E-01	1.19E-01	1.12E-01	1.01E-09	2.91E-07	9.78E-07	2.20E-01	2.78E-01	1.51E+00	2.43E-09	8.10E-07	1.67E-05
1963	3.29E-02	3.29E-02	3.29E-02	3.45E-10	8.97E-08	3.24E-07	8.76E-02	9.52E-02	8.98E-02	8.05E-10	2.33E-07	7.81E-07	1.76E-01	2.22E-01	1.21E+00	1.94E-09	6.47E-07	1.33E-05
1964	3.63E-02	3.63E-02	3.63E-02	3.81E-10	9.90E-08	3.58E-07	9.66E-02	1.05E-01	9.90E-02	8.90E-10	2.57E-07	8.63E-07	1.94E-01	2.45E-01	1.33E+00	2.15E-09	7.14E-07	1.47E-05
1965	4.58E-02	4.58E-02	4.58E-02	4.80E-10	1.25E-07	4.51E-07	1.22E-01	1.32E-01	1.25E-01	1.12E-09	3.24E-07	1.09E-06	2.45E-01	3.09E-01	1.68E+00	2.71E-09	9.01E-07	1.86E-05
1966	4.24E-02	4.24E-02	4.24E-02	4.44E-10	1.15E-07	4.18E-07	1.13E-01	1.23E-01	1.16E-01	1.04E-09	3.00E-07	1.01E-06	2.27E-01	2.86E-01	1.55E+00	2.50E-09	8.33E-07	1.72E-05
1967	5.91E-02	5.91E-02	5.91E-02	6.20E-10	1.61E-07	5.83E-07	1.57E-01	1.71E-01	1.61E-01	1.45E-09	4.19E-07	1.41E-06	3.17E-01	3.99E-01	2.17E+00	3.50E-09	1.16E-06	2.40E-05
1968	5.91E-02	5.91E-02	5.91E-02	6.20E-10	1.61E-07	5.83E-07	1.57E-01	1.71E-01	1.61E-01	1.45E-09	4.19E-07	1.41E-06	3.17E-01	3.99E-01	2.17E+00	3.50E-09	1.16E-06	2.40E-05
1969	5.91E-02	5.91E-02	5.91E-02	6.20E-10	1.61E-07	5.83E-07	1.57E-01	1.71E-01	1.61E-01	1.45E-09	4.19E-07	1.41E-06	3.17E-01	3.99E-01	2.17E+00	3.50E-09	1.16E-06	2.40E-05
1970	5.91E-02	5.91E-02	5.91E-02	6.20E-10	1.61E-07	5.83E-07	1.57E-01	1.71E-01	1.61E-01	1.45E-09	4.19E-07	1.41E-06	3.17E-01	3.99E-01	2.17E+00	3.50E-09	1.16E-06	2.40E-05
1971	5.91E-02	5.91E-02	5.91E-02	6.20E-09	6.20E-09	6.20E-09	1.57E-01	1.71E-01	1.61E-01	1.45E-08	1.61E-08	1.49E-08	3.17E-01	3.99E-01	2.17E+00	3.50E-08	4.48E-08	2.55E-07
1972	5.91E-02	5.91E-02	5.91E-02	6.20E-09	6.20E-09	6.20E-09	1.57E-01	1.71E-01	1.61E-01	1.45E-08	1.61E-08	1.49E-08	3.17E-01	3.99E-01	2.17E+00	3.50E-08	4.48E-08	2.55E-07
1973	5.91E-02	5.91E-02	5.91E-02	6.20E-09	6.20E-09	6.20E-09	1.57E-01	1.71E-01	1.61E-01	1.45E-08	1.61E-08	1.49E-08	3.17E-01	3.99E-01	2.17E+00	3.50E-08	4.48E-08	2.55E-07
1974	5.91E-02	5.91E-02	5.91E-02	1.24E-09	1.24E-09	1.24E-09	1.57E-01	1.71E-01	1.61E-01	2.90E-09	3.22E-09	2.99E-09	3.17E-01	3.99E-01	2.17E+00	6.99E-09	8.95E-09	5.10E-08
1975	5.91E-02	5.91E-02	5.91E-02	1.24E-09	1.24E-09	1.24E-09	1.57E-01	1.71E-01	1.61E-01	2.90E-09	3.22E-09	2.99E-09	3.17E-01	3.99E-01	2.17E+00	6.99E-09	8.95E-09	5.10E-08
1976	5.91E-02	5.91E-02	5.91E-02	1.24E-09	1.24E-09	1.24E-09	1.57E-01	1.71E-01	1.61E-01	2.90E-09	3.22E-09	2.99E-09	3.17E-01	3.99E-01	2.17E+00	6.99E-09	8.95E-09	5.10E-08
1977	5.91E-02	5.91E-02	5.91E-02	1.24E-09	1.24E-09	1.24E-09	1.57E-01	1.71E-01	1.61E-01	2.90E-09	3.22E-09	2.99E-09	3.17E-01	3.99E-01	2.17E+00	6.99E-09	8.95E-09	5.10E-08
1978	5.91E-02	5.91E-02	5.91E-02	1.24E-09	1.24E-09	1.24E-09	1.57E-01	1.71E-01	1.61E-01	2.90E-09	3.22E-09	2.99E-09	3.17E-01	3.99E-01	2.17E+00	6.99E-09	8.95E-09	5.10E-08
1979	5.91E-02	5.91E-02	5.91E-02	1.24E-09	1.24E-09	1.24E-09	1.57E-01	1.71E-01	1.61E-01	2.90E-09	3.22E-09	2.99E-09	3.17E-01	3.99E-01	2.17E+00	6.99E-09	8.95E-09	5.10E-08

[a] *PHED = Pesticide Handler Exposure Database.*

[b] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[c] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 6.4. *Estimates of Ontario Hydro occupational exposure (ground) to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Year	PHED[b] data—mixer/loader (ground only) total (µg/kg/day)						PHED data—right-of-way applicator total (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[c]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1948	2.90E-04	3.66E-04	1.99E-03	4.19E-11	1.40E-08	2.88E-07	3.13E-03	4.03E-03	1.21E-02	4.30E-10	1.48E-07	1.72E-06
1949	1.09E-03	1.37E-03	7.44E-03	1.47E-10	4.89E-08	1.01E-06	1.17E-02	1.51E-02	4.54E-02	1.51E-09	5.18E-07	6.01E-06
1950												
1951	1.71E-02	2.16E-02	1.17E-01	2.27E-09	7.56E-07	1.56E-05	1.85E-01	2.38E-01	7.16E-01	2.33E-08	8.02E-05	9.31E-04
1952	3.50E-02	4.42E-02	2.40E-01	4.64E-09	1.55E-06	3.18E-05	3.78E-01	4.86E-01	1.46E+00	4.76E-08	1.64E-04	1.90E-03
1953	5.70E-02	7.19E-02	3.91E-01	7.93E-09	2.64E-06	5.44E-05	6.15E-01	7.92E-01	2.38E+00	8.13E-08	2.80E-04	3.26E-03
1954	5.90E-02	7.44E-02	4.05E-01	8.73E-09	2.90E-06	5.98E-05	6.36E-01	8.19E-01	2.47E+00	8.94E-08	3.08E-04	3.57E-03
1955	8.72E-02	1.10E-01	5.98E-01	1.17E-08	3.91E-06	8.04E-05	9.40E-01	1.21E+00	3.64E+00	1.20E-07	4.14E-04	4.81E-03
1956	8.40E-02	1.06E-01	5.76E-01	1.17E-08	3.86E-06	8.00E-05	9.06E-01	1.17E+00	3.51E+00	1.20E-07	4.12E-04	4.78E-03
1957	1.40E-01	1.77E-01	9.63E-01	2.16E-08	7.18E-06	1.48E-04	1.51E+00	1.95E+00	5.87E+00	2.21E-07	7.62E-04	8.84E-03
1958	1.52E-01	1.92E-01	1.04E+00	2.35E-08	7.80E-06	1.61E-04	1.64E+00	2.11E+00	6.36E+00	2.41E-07	8.31E-04	9.64E-03
1959	1.45E-01	1.83E-01	9.93E-01	2.21E-08	7.34E-06	1.51E-04	1.56E+00	2.01E+00	6.05E+00	2.26E-07	7.79E-04	9.04E-03
1960	1.61E-01	2.03E-01	1.10E+00	2.50E-08	8.31E-06	1.71E-04	1.73E+00	2.23E+00	6.72E+00	2.56E-07	8.82E-04	1.02E-02
1961	1.78E-01	2.24E-01	1.22E+00	2.65E-08	8.83E-06	1.82E-04	1.91E+00	2.46E+00	7.42E+00	2.72E-07	9.37E-04	1.09E-02
1962	1.66E-01	2.09E-01	1.14E+00	2.44E-08	8.13E-06	1.68E-04	1.79E+00	2.30E+00	6.94E+00	2.50E-07	8.63E-04	1.00E-02
1963	1.81E-01	2.28E-01	1.24E+00	2.59E-08	8.62E-06	1.77E-04	1.95E+00	2.51E+00	7.55E+00	2.65E-07	9.14E-04	1.06E-02
1964	2.42E-01	3.05E-01	1.66E+00	3.42E-08	1.14E-05	2.35E-04	2.61E+00	3.36E+00	1.01E+01	3.51E-07	1.21E-03	1.40E-02
1965	1.84E-01	2.32E-01	1.26E+00	2.44E-08	8.11E-06	1.67E-04	1.98E+00	2.55E+00	7.68E+00	2.50E-07	8.60E-04	9.98E-03
1966	1.70E-01	2.14E-01	1.17E+00	2.25E-08	7.49E-06	1.54E-04	1.83E+00	2.36E+00	7.11E+00	2.31E-07	7.95E-04	9.22E-03
1967	2.37E-01	2.99E-01	1.63E+00	3.15E-08	1.05E-05	2.16E-04	2.56E+00	3.29E+00	9.92E+00	3.23E-07	1.11E-03	1.29E-02
1968	2.37E-01	2.99E-01	1.63E+00	3.15E-08	1.05E-05	2.16E-04	2.56E+00	3.29E+00	9.92E+00	3.23E-07	1.11E-03	1.29E-02
1969	2.37E-01	2.99E-01	1.63E+00	3.15E-08	1.05E-05	2.16E-04	2.56E+00	3.29E+00	9.92E+00	3.23E-07	1.11E-03	1.29E-02
1970	2.37E-01	2.99E-01	1.63E+00	3.15E-08	1.05E-05	2.16E-04	2.56E+00	3.29E+00	9.92E+00	3.23E-07	1.11E-03	1.29E-02
1971	2.37E-01	2.99E-01	1.63E+00	3.15E-07	4.03E-07	2.29E-06	2.56E+00	3.29E+00	9.92E+00	3.23E-06	4.27E-06	1.37E-05
1972	2.37E-01	2.99E-01	1.63E+00	3.15E-07	4.03E-07	2.29E-06	2.56E+00	3.29E+00	9.92E+00	3.23E-06	4.27E-06	1.37E-05
1973	2.37E-01	2.99E-01	1.63E+00	3.15E-07	4.03E-07	2.29E-06	2.56E+00	3.29E+00	9.92E+00	3.23E-06	4.27E-06	1.37E-05
1974	2.37E-01	2.99E-01	1.63E+00	6.29E-08	8.06E-08	4.59E-07	2.56E+00	3.29E+00	9.92E+00	6.45E-07	8.55E-07	2.74E-06
1975	2.37E-01	2.99E-01	1.63E+00	6.29E-08	8.06E-08	4.59E-07	2.56E+00	3.29E+00	9.92E+00	6.45E-07	8.55E-07	2.74E-06
1976	2.37E-01	2.99E-01	1.63E+00	6.29E-08	8.06E-08	4.59E-07	2.56E+00	3.29E+00	9.92E+00	6.45E-07	8.55E-07	2.74E-06
1977	2.37E-01	2.99E-01	1.63E+00	6.29E-08	8.06E-08	4.59E-07	2.56E+00	3.29E+00	9.92E+00	6.45E-07	8.55E-07	2.74E-06
1978	2.37E-01	2.99E-01	1.63E+00	6.29E-08	8.06E-08	4.59E-07	2.56E+00	3.29E+00	9.92E+00	6.45E-07	8.55E-07	2.74E-06
1979	2.37E-01	2.99E-01	1.63E+00	6.29E-08	8.06E-08	4.59E-07	2.56E+00	3.29E+00	9.92E+00	6.45E-07	8.55E-07	2.74E-06

[a] *Blank cells = data not available and/or no spraying occurred.*

[b] *PHED = Pesticide Handler Exposure Database.*

[c] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[d] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 6.5 *Estimates of Ministry of Natural Resources occupational exposure (aerial) to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Year	PHED[b] data—applicator—aerial, fixed wing (µg/kg/day)						PHED data—flagman—aerial, fixed wing (µg/kg/day)						PHED data—mixer/loader—aerial, fixed wing (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[c]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1953	6.62E-04	6.62E-04	6.62E-04	3.31E-11	8.61E-09	3.11E-08	1.76E-03	1.91E-03	1.81E-03	8.81E-11	2.49E-08	8.49E-08	3.54E-03	4.47E-03	2.43E-02	1.77E-10	5.81E-08	1.14E-06
1954																		
1955																		
1956	1.93E-02	1.93E-02	1.93E-02	9.64E-10	2.51E-07	9.06E-07	5.13E-02	5.58E-02	5.26E-02	2.57E-09	7.25E-07	2.47E-06	1.03E-01	1.30E-01	7.08E-01	5.16E-09	1.69E-06	3.33E-05
1957	1.13E-01	1.13E-01	1.13E-01	5.66E-09	1.47E-06	5.32E-06	3.01E-01	3.28E-01	3.09E-01	1.51E-08	4.26E-06	1.45E-05	6.06E-01	7.64E-01	4.16E+00	3.03E-08	9.94E-06	1.95E-04
1958	4.77E-02	4.77E-02	4.77E-02	2.38E-09	6.20E-07	2.24E-06	1.27E-01	1.38E-01	1.30E-01	6.35E-09	1.79E-06	6.11E-06	2.55E-01	3.22E-01	1.75E+00	1.28E-08	4.18E-06	8.22E-05
1959	7.48E-02	7.48E-02	7.48E-02	3.74E-09	9.72E-07	3.51E-06	1.99E-01	2.16E-01	2.04E-01	9.95E-09	2.81E-06	9.58E-06	4.00E-01	5.05E-01	2.74E+00	2.00E-08	6.56E-06	1.29E-04
1960	4.10E-02	4.10E-02	4.10E-02	2.05E-09	5.33E-07	1.93E-06	1.09E-01	1.19E-01	1.12E-01	5.45E-09	1.54E-06	5.25E-06	2.19E-01	2.77E-01	1.50E+00	1.10E-08	3.60E-06	7.07E-05
1961	8.52E-02	8.52E-02	8.52E-02	4.26E-09	1.11E-06	4.00E-06	2.27E-01	2.46E-01	2.32E-01	1.13E-08	3.20E-06	1.09E-05	4.56E-01	5.75E-01	3.13E+00	2.28E-08	7.47E-06	1.47E-04
1962	3.61E-02	3.61E-02	3.61E-02	1.81E-09	4.70E-07	1.70E-06	9.62E-02	1.04E-01	9.85E-02	4.81E-09	1.36E-06	4.63E-06	1.93E-01	2.44E-01	1.33E+00	9.67E-09	3.17E-06	6.23E-05
1963	9.42E-02	9.42E-02	9.42E-02	4.71E-09	1.22E-06	4.43E-06	2.51E-01	2.72E-01	2.57E-01	1.25E-08	3.54E-06	1.21E-05	5.04E-01	6.36E-01	3.46E+00	2.52E-08	8.27E-06	1.62E-04
1964	9.54E-02	9.54E-02	9.54E-02	4.77E-09	1.24E-06	4.49E-06	2.54E-01	2.76E-01	2.60E-01	1.27E-08	3.59E-06	1.22E-05	5.11E-01	6.44E-01	3.50E+00	2.55E-08	8.37E-06	1.65E-04
1965	3.95E-02	3.95E-02	3.95E-02	1.97E-09	5.13E-07	1.86E-06	1.05E-01	1.14E-01	1.08E-01	5.26E-09	1.49E-06	5.06E-06	2.11E-01	2.67E-01	1.45E+00	1.06E-08	3.47E-06	6.81E-05
1966	1.02E-02	1.02E-02	1.02E-02	5.08E-10	1.32E-07	4.77E-07	2.70E-02	2.94E-02	2.77E-02	1.35E-09	3.82E-07	1.30E-06	5.43E-02	6.85E-02	3.72E-01	2.72E-09	8.91E-07	1.75E-05
1967	2.08E-02	2.08E-02	2.08E-02	1.04E-09	2.70E-07	9.77E-07	5.53E-02	6.01E-02	5.67E-02	2.77E-09	7.81E-07	2.67E-06	1.11E-01	1.40E-01	7.63E-01	5.56E-09	1.82E-06	3.58E-05
1968																		
1969	1.37E-02	1.37E-02	1.37E-02	6.84E-10	1.78E-07	6.43E-07	3.64E-02	3.96E-02	3.73E-02	1.82E-09	5.14E-07	1.75E-06	7.33E-02	9.23E-02	5.02E-01	3.66E-09	1.20E-06	2.36E-05
1970																		
1971																		
1972																		
1973	6.53E-03	6.53E-03	6.53E-03	3.27E-09	3.27E-09	3.27E-09	1.74E-02	1.89E-02	1.78E-02	8.69E-09	9.44E-09	8.91E-09	3.50E-02	4.41E-02	2.40E-01	1.75E-08	2.20E-08	1.20E-07
1974																		
1975																		
1976																		
1977																		
1978																		
1979	2.65E-03	2.65E-03	2.65E-03	2.65E-10	2.65E-10	2.65E-10	7.05E-03	7.66E-03	7.22E-03	7.05E-10	7.66E-10	7.22E-10	1.42E-02	1.79E-02	9.72E-02	1.42E-09	1.79E-09	9.72E-09

[a] *Blank cells = data not available and/or no spraying occurred.*
[b] *PHED =Pesticide Handler Exposure Database.*
[c] *Low, ce ntral, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[d] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 6.6. *Estimates of Ministry of Natural Resources occupational exposure (ground-vehicle) to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Year	PHED[b] data—mixer/loader (ground only) total (µg/kg/day)						PHED data—right-of-way applicator total (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[c]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1959	1.35E-03	1.70E-03	9.24E-03	6.74E-11	2.21E-08	4.34E-07	1.45E-02	1.87E-02	5.64E-02	7.27E-10	2.43E-07	2.65E-06[d]
1960												
1961												
1962												
1963												
1964												
1965												
1966												
1967												
1968												
1969												
1970												
1971	2.46E-04	3.10E-04	1.69E-03	2.46E-11	3.10E-11	1.69E-10	2.65E-03	3.42E-03	1.03E-02	2.65E-10	3.42E-10	1.03E-09
1972												
1973												
1974												
1975												
1976												
1977												
1978												
1979												

[a] *Blank cells = data not available and/or no spraying occurred.*

[b] *PHED = Pesticide Handler Exposure Database.*

[c] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[d] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 6.7. *Estimates of Ministry of Natural Resources occupational exposure (ground-backpack) to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Year	PHED[b] data—mixer/loader (ground only) total (µg/kg/day)						PHED data—right-of-way applicator total (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[c]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1948	3.77E-03	7.52E-03	7.52E-03	1.88E-10	9.77E-08	3.53E-07	4.46E-03	8.90E-03	8.90E-03	2.23E-10	1.16E-07	4.18E-07
1949												
1950												
1951												
1952												
1953	5.30E-01	1.06E+00	1.06E+00	2.65E-08	1.37E-05[d]	4.97E-05	6.28E-01	1.25E+00	1.25E+00	3.14E-08	1.63E-05	5.89E-05
1954												
1955												
1956												
1957	6.88E-01	1.37E+00	1.37E+00	3.44E-08	1.79E-05	6.45E-05	8.15E-01	1.63E+00	1.63E+00	4.08E-08	2.11E-05	7.64E-05
1958	7.54E-01	1.50E+00	1.50E+00	3.77E-08	1.95E-05	7.07E-05	8.93E-01	1.78E+00	1.78E+00	4.46E-08	2.31E-05	8.37E-05
1959	1.01E+00	2.01E+00	2.01E+00	5.03E-08	2.61E-05	9.43E-05	1.19E+00	2.38E+00	2.38E+00	5.96E-08	3.09E-05	1.12E-04
1960	9.90E-01	1.97E+00	1.97E+00	4.95E-08	2.57E-05	9.28E-05	1.17E+00	2.34E+00	2.34E+00	5.86E-08	3.04E-05	1.10E-04
1961	2.46E-01	4.91E-01	4.91E-01	1.23E-08	6.39E-06	2.31E-05	2.92E-01	5.82E-01	5.82E-01	1.46E-08	7.56E-06	2.73E-05
1962	2.10E+00	4.19E+00	4.19E+00	1.05E-07	5.44E-05	1.97E-04	2.48E+00	4.96E+00	4.96E+00	1.24E-07	6.44E-05	2.33E-04
1963	1.07E-01	2.13E-01	2.13E-01	5.34E-09	2.77E-06	1.00E-05	1.26E-01	2.52E-01	2.52E-01	6.32E-09	3.28E-06	1.19E-05
1964	1.70E+00	3.39E+00	3.39E+00	8.49E-08	4.40E-05	1.59E-04	2.01E+00	4.01E+00	4.01E+00	1.00E-07	5.21E-05	1.88E-04
1965	7.32E-01	1.46E+00	1.46E+00	3.66E-08	1.90E-05	6.87E-05	8.67E-01	1.73E+00	1.73E+00	4.34E-08	2.25E-05	8.13E-05
1966	2.26E-01	4.51E-01	4.51E-01	1.13E-08	5.86E-06	2.12E-05	2.68E-01	5.34E-01	5.34E-01	1.34E-08	6.94E-06	2.51E-05
1967	1.05E+00	2.09E+00	2.09E+00	5.23E-07	1.04E-06	1.04E-06	1.24E+00	2.47E+00	2.47E+00	6.20E-07	1.24E-06	1.24E-06
1968	4.52E-01	9.02E-01	9.02E-01	2.26E-07	4.51E-07	4.51E-07	5.36E-01	1.07E+00	1.07E+00	2.68E-07	5.34E-07	5.34E-07
1969												
1970												
1971												
1972												
1973	5.53E-02	1.10E-01	1.10E-01	5.53E-09	1.10E-08	1.10E-08	6.55E-02	1.31E-01	1.31E-01	6.55E-09	1.31E-08	1.31E-08
1974	1.13E-01	2.26E-01	2.26E-01	1.13E-08	2.26E-08	2.26E-08	1.34E-01	2.67E-01	2.67E-01	1.34E-08	2.67E-08	2.67E-08

[a] *Blank cells = data not available and/or no spraying occurred.*

[b] *PHED = Pesticide Handler Exposure Database.*

[c] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[d] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 6.8. Estimate of recreational visitor exposures to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).

	Unit	Ontario Hydro				MNR			
		2,4,5-T	TCDD			2,4,5-T	TCDD		
			Low[a]	Central	High		Low	Central	High
Receptor parameter									
Body weight	kg	70.7	70.7	70.7	70.7	70.7	70.7	70.7	70.7
Amount of soil consumed	mg/day	50	50	50	50	50	50	50	50
Exposed surface area	cm^2	4343	4343	4343	4343	4343	4343	4343	4343
Soil adherence factor—body	g/cm^2 / event	0.00007	0.00007	0.00007	0.00007	0.00007	0.00007	0.00007	0.00007
Inhalation rate	m^3/day	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6
Particulate concentration in air	g/m^3	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07
Hours per day	hr	4	4	4	4	4	4	4	4
Days per year (direct soil)	d/yr	14	14	14	14	14	14	14	14
Averaging time	d/yr	365	365	365	365	365	365	365	365
Wild berry consumption rate	g/day	5.2	5.2	5.2	5.2	5.2	5.2	5.2	5.2
Local fish consumption rate	g/day	24.42	24.42	24.42	24.42	24.42	24.42	24.42	24.42
Wild game consumption rate	g/day	1.8	1.8	1.8	1.8	1.7	1.7	1.7	1.7
Chemical parameter									
Soil concentration—ingestion/ inhalation/dermal	µg/g	9.41E-01	4.72E-07	1.49E-05	6.43E-05	4.03E-01	2.02E-08	5.25E-06	1.90E-05
Wild berry concentration	µg/g	4.36E-03	2.07E-11	6.51E-10	2.82E-09	1.87E-03	8.84E-13	2.30E-10	8.31E-10
Fish concentration	µg/g	NA[b]	9.66E-10	2.51E-07	9.08E-07	NA	4.14E-10	1.08E-07	3.89E-07
Wild game concentration	µg/g	NA	4.03E-10	1.5E-08	6.3E-08	NA	2.11E-11	5.48E-09	1.98E-08
Soil bioavailability	unitless	1	0.34	0.34	0.34	1	0.34	0.34	0.34
Dermal RAF	unitless	0.03	0.08	0.08	0.08	0.03	0.08	0.08	0.08
Exposure estimate									
Soil ingestion	µg/kg/day	2.55E-05	4.36E-12	1.37E-10	5.93E-10	1.09E-05	1.86E-13	4.84E-11	1.75E-10
Soil inhalation	µg/kg/day	6.44E-09	3.23E-15	1.02E-13	4.40E-13	2.76E-09	1.38E-16	3.59E-14	1.30E-13
Soil dermal	µg/kg/day	3.95E-06	5.86E-12	1.84E-10	7.98E-10	1.69E-06	2.50E-13	6.51E-11	2.35E-10
Wild berry	µg/kg/day	3.21E-04	1.52E-12	4.79E-11	2.07E-10	1.37E-04	6.50E-14	1.69E-11	6.11E-11
Fish and wild game	µg/kg/day	NA	3.85E-11	8.87E-09	3.22E-08	NA	1.46E-11	3.79E-09	1.37E-08
Exposure									
Recreational visitor (adult)	µg/kg/day	3.50E-04	1.17E-11	3.70E-10	1.60E-09	1.50E-04	5.02E-13	1.30E-10	4.71E-10
Hunter/angler (total)	µg/kg/day	NA	5.02E-11	9.24E-09	3.38E-08	NA	1.51E-11	3.92E-09	1.42E-08

[a] Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.

[b] NA = not applicable.

Table 6.9. *Estimates of residential exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

	Unit	Ontario Hydro				Ministry of Transportation			
		2,4,5-T	TCDD			2,4,5-T	TCDD		
			Low[a]	Central	High		Low	Central	High
Receptor parameter									
Body weight (toddler)	kg	16.5	16.5	16.5	16.5	16.5	16.5	16.5	16.5
Amount of soil consumed	mg/day	200	200	200	200	200	200	200	200
Exposed surface area	cm2	1745	1745	1745	1745	1745	1745	1745	1745
Soil adherence factor—body	g/cm^2 / event	0.0002	0.0002	0.0002	0.0002	0.0002	0.0002	0.0002	0.0002
Inhalation rate	m3/day	8.3	8.3	8.3	8.3	8.3	8.3	8.3	8.3
Particulate concentration in air	g/m3	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07	7.60E-07
Hours per day	hr	24	24	24	24	24	24	24	24
Days per year (direct soil)	d/yr	275	275	275	275	275	275	275	275
Averaging time	d/yr	365	365	365	365	365	365	365	365
Wild berry consumption rate	g/day	1.2	1.2	1.2	1.2	1.2	1.2	1.2	1.2
Local fish consumption rate	g/day	21.3	21.3	21.3	21.3	21.3	21.3	21.3	21.3
Wild game consumption rate	g/day	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8
Chemical parameter									
Soil concentration—ingestion/inhalation/ dermal	µg/g	5.23E-01	2.62E-07	8.26E-06	3.58E-05	4.51E-01	4.00E-07	2.00E-05	1.01E-04
Wild berry concentration	µg/g	2.42E-03	1.15E-11	3.62E-10	1.57E-09	2.09E-03	1.75E-11	8.74E-10	4.41E-09
Fish concentration	µg/g	NA[b]	9.66E-10	2.51E-07	9.08E-07	NA	2.49E-09	6.47E-07	2.34E-06
Wild game concentration	µg/g	NA	4.03E-10	1.50E-08	6.30E-08	NA			
Soil bioavailability	unitless	1	0.34	0.34	0.34	1	0.34	0.34	0.34
Dermal RAF	unitless	0.14	0.37	0.37	0.37	0.14	0.37	0.37	0.37
Exposure estimate									
Soil ingestion	µg/kg/day	4.78E-03	8.15E-10	2.56E-08	1.11E-07	4.12E-03	1.24E-09	6.20E-08	3.13E-07
Soil inhalation	µg/kg/day	1.51E-07	7.56E-14	2.38E-12	1.03E-11	1.30E-07	1.15E-13	5.75E-12	2.90E-11
Soil dermal	µg/kg/day	1.19E-03	1.57E-09	4.93E-08	2.14E-07	1.03E-03	2.39E-09	1.19E-07	6.01E-07
Wild berry	µg/kg/day	1.76E-04	8.36E-13	2.63E-11	1.14E-10	1.52E-04	1.27E-12	6.36E-11	3.21E-10
Fish and wild game	µg/kg/day	NA	1.35E-10	3.28E-08	1.19E-07	NA	3.21E-10	8.35E-08	3.02E-07
Exposure									
Resident (toddler)	µg/kg/day	6.15E-03	2.38E-09	7.50E-08	3.25E-07	5.30E-03	3.63E-09	1.81E-07	9.14E-07
Hunter/angler child (toddler)	µg/kg/day	NA	2.52E-09	1.08E-07	4.43E-07	NA	3.95E-09	2.65E-07	1.22E-06

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[b] *NA = not applicable.*

Table 6.10. *Estimates of fish consumption exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (µg/kg/day).*

Receptor	Ontario Hydro			Ministry of Natural Resources		
	Low[a]	Central	High	Low	Central	High
Angler-fish consumer	3.3E-11	8.7E-09	3.1E-08	1.4E-11	3.7E-09	1.3E-08
First Nations-fish consumer	7.5E-10	2.0E-07	7.1E-07	3.2E-10	8.4E-08	3.0E-07

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

Table 6.11. *Estimates of wild game consumption exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (µg/kg/day).*

Ontario Hydro	Moose			White-tailed deer		
	Low[a]	Central	High	Low	Central	High
Hunter-game consumer	5.1E-11	1.9E-09	8.0E-09	2.2E-11	1.3E-09	5.0E-09
First Nations-game consumer	7.7E-10	2.9E-08	1.2E-07	3.4E-10	1.9E-08	7.5E-08
Ministry of Natural Resources	**Moose**			**White-tailed deer**		
	Low	Central	High	Low	Central	High
Hunter-game consumer	2.7E-12	7.0E-10	2.5E-09	1.9E-12	4.9E-10	1.8E-09
First Nations-game consumer	4.0E-11	1.0E-08	3.8E-08	2.8E-11	7.4E-09	2.7E-08

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

Table 6.12. *Estimates of bystander exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)—1 day per year direct spray (Ontario Hydro).*

Year	Ground-based exposure at 30 m (µg/kg/day)				Aerial (fixed)-based exposure at 500 m (µg/kg/day)				Aerial (rotary)-based exposure at 500 m (µg/kg/day)			
	2,4,5-T	TCDD			2,4,5-T	TCDD			2,4,5-T	TCDD		
		Low[a]	Central	High		Low	Central	High		Low	Central	High
1948	4.91E+00	3.26E-07	8.48E-05	3.06E-04[b]	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1949	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1950	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1951	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1952	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1953	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1954	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1955	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1956	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1957	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1958	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1959	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1960	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1961	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1962	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1963	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1964	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1965	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1966	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1967	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1968	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1969	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1970	4.91E+00	3.26E-07	8.48E-05	3.06E-04	1.23E+00	2.57E-08	1.60E-05	8.69E-05	2.45E+00	1.63E-07	4.24E-05	1.53E-04
1971	4.91E+00	3.26E-06	3.26E-06	3.26E-06	1.23E+00	2.57E-07	6.17E-07	9.25E-07	2.45E+00	1.63E-06	1.63E-06	1.63E-06
1972	4.91E+00	3.26E-06	3.26E-06	3.26E-06	1.23E+00	2.57E-07	6.17E-07	9.25E-07	2.45E+00	1.63E-06	1.63E-06	1.63E-06
1973	4.91E+00	3.26E-06	3.26E-06	3.26E-06	1.23E+00	2.57E-07	6.17E-07	9.25E-07	2.45E+00	1.63E-06	1.63E-06	1.63E-06
1974	4.91E+00	6.52E-07	6.52E-07	6.52E-07	1.23E+00	5.14E-08	1.23E-07	1.85E-07	2.45E+00	3.26E-07	3.26E-07	3.26E-07
1975	4.91E+00	6.52E-07	6.52E-07	6.52E-07	1.23E+00	5.14E-08	1.23E-07	1.85E-07	2.45E+00	3.26E-07	3.26E-07	3.26E-07
1976	4.91E+00	6.52E-07	6.52E-07	6.52E-07	1.23E+00	5.14E-08	1.23E-07	1.85E-07	2.45E+00	3.26E-07	3.26E-07	3.26E-07
1977	4.91E+00	6.52E-07	6.52E-07	6.52E-07	1.23E+00	5.14E-08	1.23E-07	1.85E-07	2.45E+00	3.26E-07	3.26E-07	3.26E-07
1978	4.91E+00	6.52E-07	6.52E-07	6.52E-07	1.23E+00	5.14E-08	1.23E-07	1.85E-07	2.45E+00	3.26E-07	3.26E-07	3.26E-07
1979	4.91E+00	6.52E-07	6.52E-07	6.52E-07	1.23E+00	5.14E-08	1.23E-07	1.85E-07	2.45E+00	3.26E-07	3.26E-07	3.26E-07

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[b] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 6.13. *Estimates of bystander exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)—1 day per year direct spray.*

Year	Ground-based exposure at 30 m (µg/kg/day) Ministry of Transportation				Aerial (fixed)-based exposure at 500 m (µg/kg/day) Ministry of Natural Resources			
	2,4,5-T	TCDD			2,4,5-T	TCDD		
		Low[a]	Central	High		Low	Central	High
1953	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04[b]
1954	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1955	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1956	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1957	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1958	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1959	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1960	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1961	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1962	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1963	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1964	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1965	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1966	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1967	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1968	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1969	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1970	2.12E+00	1.41E-07	3.65E-05	1.32E-04	5.26E+00	3.49E-07	9.08E-05	3.28E-04
1971	2.12E+00	1.41E-06	1.41E-06	1.41E-06	5.26E+00	3.49E-06	3.49E-06	3.49E-06
1972	2.12E+00	1.41E-06	1.41E-06	1.41E-06	5.26E+00	3.49E-06	3.49E-06	3.49E-06
1973	2.12E+00	1.41E-06	1.41E-06	1.41E-06	5.26E+00	3.49E-06	3.49E-06	3.49E-06

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[b] *Coloured font = exposure estimates greater than the relevant toxicological reference values defined in Chapter 3 of this report.*

Table 6.14. *Estimates of Ministry of Transportation occupational margins of safety for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Year	PHED[a] data—mixer/loader total (µg/kg/day)						PHED data—right-of-way applicator total (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[b]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1953	0.0[c]	0.0	0.0	0.0	0.8	15.9[d]	0.0	0.1	0.2	0.0	8.2	95.3
1954	0.0	0.0	0.0	0.0	0.8	16.0	0.0	0.1	0.2	0.0	8.2	95.6
1955	0.0	0.0	0.0	0.0	0.8	16.4	0.0	0.1	0.2	0.0	8.4	97.9
1956	0.0	0.0	0.0	0.0	0.8	16.3	0.0	0.1	0.2	0.0	8.4	97.1
1957	0.0	0.0	0.0	0.0	0.8	16.8	0.0	0.1	0.2	0.0	8.7	100.5
1958	0.0	0.0	0.0	0.0	0.6	12.3	0.0	0.0	0.1	0.0	6.3	73.7
1959	0.0	0.0	0.0	0.0	0.7	13.5	0.0	0.0	0.1	0.0	7.0	80.8
1960	0.0	0.0	0.0	0.0	0.7	15.1	0.0	0.1	0.2	0.0	7.8	90.2
1961	0.0	0.0	0.0	0.0	0.8	15.6	0.0	0.1	0.2	0.0	8.0	93.2
1962	0.0	0.0	0.0	0.0	0.9	17.8	0.0	0.1	0.2	0.0	9.2	106.5
1963	0.0	0.0	0.0	0.0	1.1	23.3	0.1	0.1	0.2	0.0	12.0	139.1
1964	0.0	0.0	0.0	0.0	1.2	24.8	0.1	0.1	0.3	0.0	12.8	148.4
1965	0.0	0.0	0.0	0.0	1.1	22.0	0.1	0.1	0.2	0.0	11.4	131.7
1966	0.0	0.0	0.0	0.0	1.2	25.0	0.1	0.1	0.3	0.0	12.9	149.4
1967	0.0	0.0	0.1	0.0	1.5	31.9	0.1	0.1	0.3	0.0	16.4	190.6
1968	0.0	0.0	0.1	0.0	1.5	30.9	0.1	0.1	0.3	0.0	15.9	184.5
1969	0.0	0.0	0.1	0.0	1.5	30.9	0.1	0.1	0.3	0.0	15.9	184.4
1970	0.0	0.0	0.1	0.0	1.6	33.2	0.1	0.1	0.4	0.0	17.1	198.3
1971	0.0	0.0	0.0	0.0	0.1	0.3	0.1	0.1	0.3	0.4	0.6	1.8
1972	0.0	0.0	0.1	0.0	0.1	0.3	0.1	0.1	0.3	0.5	0.6	2.0
1973	0.0	0.0	0.1	0.0	0.1	0.3	0.1	0.1	0.3	0.5	0.6	2.0

[a] *PHED = Pesticide Handler Exposure Database.*

[b] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*

[c] *The value 0.0 = <0.05 due to rounding.*

[d] *Coloured font = exposure exceeded the toxicological reference value (defined in Chapter 3).*

Table 6.15. *Estimates of Ontario Hydro occupational (aerial) margins of safety for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*

Year	PHED[a] data—applicator—aerial, fixed wing (µg/kg/day)						PHED data—flagman—aerial, fixed wing (µg/kg/day)						PHED data—mixer/loader—aerial, fixed wing (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[b]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1953	0.0[c]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.1	1.1[d]
1954	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.1	0.0	0.1	2.7
1955	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.5
1956	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.1	1.6
1957	0.0	0.0	0.0	0.0	0.1	0.2	0.0	0.0	0.0	0.0	0.2	0.5	0.0	0.0	0.2	0.0	0.4	8.9
1958	0.0	0.0	0.0	0.0	0.1	0.2	0.0	0.0	0.0	0.0	0.2	0.6	0.0	0.0	0.2	0.0	0.5	10.0
1959	0.0	0.0	0.0	0.0	0.1	0.2	0.0	0.0	0.0	0.0	0.1	0.5	0.0	0.0	0.2	0.0	0.4	8.5
1960	0.0	0.0	0.0	0.0	0.1	0.3	0.0	0.0	0.0	0.0	0.2	0.6	0.0	0.0	0.2	0.0	0.5	10.9
1961	0.0	0.0	0.0	0.0	0.1	0.2	0.0	0.0	0.0	0.0	0.2	0.5	0.0	0.0	0.2	0.0	0.4	8.9
1962	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.1	0.4	0.0	0.0	0.2	0.0	0.4	7.3
1963	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.3	0.0	0.0	0.1	0.0	0.3	5.8
1964	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.1	0.4	0.0	0.0	0.1	0.0	0.3	6.4
1965	0.0	0.0	0.0	0.0	0.1	0.2	0.0	0.0	0.0	0.0	0.1	0.5	0.0	0.0	0.2	0.0	0.4	8.1
1966	0.0	0.0	0.0	0.0	0.1	0.2	0.0	0.0	0.0	0.0	0.1	0.4	0.0	0.0	0.2	0.0	0.4	7.5
1967	0.0	0.0	0.0	0.0	0.1	0.3	0.0	0.0	0.0	0.0	0.2	0.6	0.0	0.0	0.2	0.0	0.5	10.4
1968	0.0	0.0	0.0	0.0	0.1	0.3	0.0	0.0	0.0	0.0	0.2	0.6	0.0	0.0	0.2	0.0	0.5	10.4
1969	0.0	0.0	0.0	0.0	0.1	0.3	0.0	0.0	0.0	0.0	0.2	0.6	0.0	0.0	0.2	0.0	0.5	10.4
1970	0.0	0.0	0.0	0.0	0.1	0.3	0.0	0.0	0.0	0.0	0.2	0.6	0.0	0.0	0.2	0.0	0.5	10.4
1971	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.1
1972	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.1
1973	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.1
1974	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0
1975	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0
1976	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0
1977	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0
1978	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0
1979	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0

[a] *PHED = Pesticide Handler Exposure Database.*
[b] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[c] *The value 0.0 = <0.05 due to rounding.*
[d] *Coloured font = exposure exceeded the TRV (TDI).*

Table 6.16. *Estimates of Ontario Hydro occupational (ground) margins of safety for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Year	PHED[b] data—mixer/loader (ground only) total (µg/kg/day)						PHED data—right-of-way applicator total (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[c]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1948	0.0[d]	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.7
1949	0.0	0.0	0.0	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.2	2.6[e]
1950												
1951	0.0	0.0	0.0	0.0	0.3	6.8	0.0	0.0	0.1	0.0	3.5	40.5
1952	0.0	0.0	0.0	0.0	0.7	13.8	0.0	0.0	0.1	0.0	7.1	82.7
1953	0.0	0.0	0.0	0.0	1.1	23.6	0.1	0.1	0.2	0.0	12.2	141.2
1954	0.0	0.0	0.0	0.0	1.3	26.0	0.1	0.1	0.2	0.0	13.4	155.4
1955	0.0	0.0	0.1	0.0	1.7	35.0	0.1	0.1	0.4	0.1	18.0	209.0
1956	0.0	0.0	0.1	0.0	1.7	34.8	0.1	0.1	0.4	0.1	17.9	208.0
1957	0.0	0.0	0.1	0.0	3.1	64.3	0.2	0.2	0.6	0.1	33.1	384.4
1958	0.0	0.0	0.1	0.0	3.4	70.1	0.2	0.2	0.6	0.1	36.1	419.2
1959	0.0	0.0	0.1	0.0	3.2	65.8	0.2	0.2	0.6	0.1	33.9	392.9
1960	0.0	0.0	0.1	0.0	3.6	74.4	0.2	0.2	0.7	0.1	38.3	444.7
1961	0.0	0.0	0.1	0.0	3.8	79.1	0.2	0.2	0.7	0.1	40.7	472.5
1962	0.0	0.0	0.1	0.0	3.5	72.8	0.2	0.2	0.7	0.1	37.5	435.2
1963	0.0	0.0	0.1	0.0	3.7	77.2	0.2	0.3	0.8	0.1	39.7	461.0
1964	0.0	0.0	0.2	0.0	5.0	102.0	0.3	0.3	1.0	0.2	52.5	609.3
1965	0.0	0.0	0.1	0.0	3.5	72.6	0.2	0.3	0.8	0.1	37.4	433.7
1966	0.0	0.0	0.1	0.0	3.3	67.1	0.2	0.2	0.7	0.1	34.6	400.9
1967	0.0	0.0	0.2	0.0	4.6	93.8	0.3	0.3	1.0	0.1	48.3	560.4
1968	0.0	0.0	0.2	0.0	4.6	93.8	0.3	0.3	1.0	0.1	48.3	560.4
1969	0.0	0.0	0.2	0.0	4.6	93.8	0.3	0.3	1.0	0.1	48.3	560.4
1970	0.0	0.0	0.2	0.0	4.6	93.8	0.3	0.3	1.0	0.1	48.3	560.4
1971	0.0	0.0	0.2	0.1	0.2	1.0	0.3	0.3	1.0	1.4	1.9	6.0
1972	0.0	0.0	0.2	0.1	0.2	1.0	0.3	0.3	1.0	1.4	1.9	6.0
1973	0.0	0.0	0.2	0.1	0.2	1.0	0.3	0.3	1.0	1.4	1.9	6.0
1974	0.0	0.0	0.2	0.0	0.0	0.2	0.3	0.3	1.0	0.3	0.4	1.2
1975	0.0	0.0	0.2	0.0	0.0	0.2	0.3	0.3	1.0	0.3	0.4	1.2
1976	0.0	0.0	0.2	0.0	0.0	0.2	0.3	0.3	1.0	0.3	0.4	1.2
1977	0.0	0.0	0.2	0.0	0.0	0.2	0.3	0.3	1.0	0.3	0.4	1.2
1978	0.0	0.0	0.2	0.0	0.0	0.2	0.3	0.3	1.0	0.3	0.4	1.2
1979	0.0	0.0	0.2	0.0	0.0	0.2	0.3	0.3	1.0	0.3	0.4	1.2

[a] *Blank cells = Information not available.*
[b] *PHED = Pesticide Handler Exposure Database.*
[c] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[d] *The value 0.0 = <0.05 due to rounding.*
[e] *Coloured font = exposure exceeded the TRV (TDI).*

Table 6.17. *Estimates of Ministry of Natural Resources occupational (aerial) margins of safety for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Year	PHED[b] data—applicator—aerial, fixed wing (µg/kg/day)						PHED data—flagman—aerial, fixed wing (µg/kg/day)						PHED data—mixer/loader—aerial, fixed wing (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[c]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1952																		
1953	0.0[d]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.5
1954																		
1955																		
1956	0.0	0.0	0.0	0.0	0.1	0.4	0.0	0.0	0.0	0.0	0.3	1.1[e]	0.0	0.0	0.1	0.0	0.7	14.5
1957	0.0	0.0	0.0	0.0	0.6	2.3	0.0	0.0	0.0	0.0	1.9	6.3	0.1	0.1	0.4	0.0	4.3	84.9
1958	0.0	0.0	0.0	0.0	0.3	1.0	0.0	0.0	0.0	0.0	0.8	2.7	0.0	0.0	0.2	0.0	1.8	35.8
1959	0.0	0.0	0.0	0.0	0.4	1.5	0.0	0.0	0.0	0.0	1.2	4.2	0.0	0.1	0.3	0.0	2.9	56.1
1960	0.0	0.0	0.0	0.0	0.2	0.8	0.0	0.0	0.0	0.0	0.7	2.3	0.0	0.0	0.2	0.0	1.6	30.7
1961	0.0	0.0	0.0	0.0	0.5	1.7	0.0	0.0	0.0	0.0	1.4	4.7	0.0	0.1	0.3	0.0	3.2	63.9
1962	0.0	0.0	0.0	0.0	0.2	0.7	0.0	0.0	0.0	0.0	0.6	2.0	0.0	0.0	0.1	0.0	1.4	27.1
1963	0.0	0.0	0.0	0.0	0.5	1.9	0.0	0.0	0.0	0.0	1.5	5.3	0.1	0.1	0.3	0.0	3.6	70.6
1964	0.0	0.0	0.0	0.0	0.5	2.0	0.0	0.0	0.0	0.0	1.6	5.3	0.1	0.1	0.4	0.0	3.6	71.6
1965	0.0	0.0	0.0	0.0	0.2	0.8	0.0	0.0	0.0	0.0	0.6	2.2	0.0	0.0	0.1	0.0	1.5	29.6
1966	0.0	0.0	0.0	0.0	0.1	0.2	0.0	0.0	0.0	0.0	0.2	0.6	0.0	0.0	0.0	0.0	0.4	7.6
1967	0.0	0.0	0.0	0.0	0.1	0.4	0.0	0.0	0.0	0.0	0.3	1.2	0.0	0.0	0.1	0.0	0.8	15.6
1968																		
1969	0.0	0.0	0.0	0.0	0.1	0.3	0.0	0.0	0.0	0.0	0.2	0.8	0.0	0.0	0.1	0.0	0.5	10.3
1970																		
1971																		
1972																		
1973	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1
1974																		
1975																		
1976																		
1977																		
1978																		
1979	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

[a] *Blank cells = Information not available.*
[b] *PHED = Pesticide Handler Exposure Database.*
[c] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[d] *The value 0.0 = <0.05 due to rounding.*
[e] *Coloured font = exposure exceeded the TRV (TDI).*

Table 6.18. *Estimates of Ministry of Natural Resources occupational (ground-vehicle) margins of safety for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Year	PHED[b] data—mixer/loader (ground only) total (µg/kg/day)						PHED data—right-of-way applicator total (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[c]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1950												
1951												
1952												
1953												
1954												
1955												
1956												
1957												
1958												
1959	0.0[d]	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.1	1.2[e]
1960												
1961												
1962												
1963												
1964												
1965												
1966												
1967												
1968												
1969												
1970												
1971	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1972												
1973												
1974												
1975												
1976												
1977												
1978												
1979												

[a] *Blank cells = Information not available.*
[b] *PHED = Pesticide Handler Exposure Database.*
[c] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[d] *The value 0.0 = <0.05 due to rounding.*
[e] *Coloured font = exposure exceeded the TRV (TDI).*

Table 6.19. *Estimates of Ministry of Natural Resources occupational (ground-backpack) margins of safety for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Year	PHED[b] data—mixer/loader (ground only) total (µg/kg/day)						PHED data—right-of-way applicator total (µg/kg/day)					
	2,4,5-T			TCDD			2,4,5-T			TCDD		
	Low[c]	Central	High	Low	Central	High	Low	Central	High	Low	Central	High
1948	0.0[d]	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.1	0.2
1949												
1950												
1951												
1952												
1953	0.1	0.1	0.1	0.0	6.0[e]	21.6	0.1	0.1	0.1	0.0	7.1	25.6
1954												
1955												
1956												
1957	0.1	0.1	0.1	0.0	7.8	28.1	0.1	0.2	0.2	0.0	9.2	33.2
1958	0.1	0.2	0.2	0.0	8.5	30.7	0.1	0.2	0.2	0.0	10.1	36.4
1959	0.1	0.2	0.2	0.0	11.3	41.0	0.1	0.2	0.2	0.0	13.4	48.6
1960	0.1	0.2	0.2	0.0	11.2	40.4	0.1	0.2	0.2	0.0	13.2	47.8
1961	0.0	0.0	0.0	0.0	2.8	10.0	0.0	0.1	0.1	0.0	3.3	11.9
1962	0.2	0.4	0.4	0.0	23.7	85.5	0.2	0.5	0.5	0.1	28.0	101.3
1963	0.0	0.0	0.0	0.0	1.2	4.4	0.0	0.0	0.0	0.0	1.4	5.2
1964	0.2	0.3	0.3	0.0	19.1	69.2	0.2	0.4	0.4	0.0	22.7	81.9
1965	0.1	0.1	0.1	0.0	8.3	29.9	0.1	0.2	0.2	0.0	9.8	35.4
1966	0.0	0.0	0.0	0.0	2.5	9.2	0.0	0.1	0.1	0.0	3.0	10.9
1967	0.1	0.2	0.2	0.2	0.5	0.5	0.1	0.2	0.2	0.3	0.5	0.5
1968	0.0	0.1	0.1	0.1	0.2	0.2	0.1	0.1	0.1	0.1	0.2	0.2
1969												
1970												
1971												
1972												
1973	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1974	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1975	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.1	0.2
1976												
1977												
1978												

[a] *Blank cells = information not available.*
[b] *PHED = Pesticide Handler Exposure Database.*
[c] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[d] *The value 0.0 = <0.05 due to rounding.*
[e] *Coloured font = exposure exceeded the TRV (TDI).*

Table 6.20. *Estimates of recreational visitor margins of safety for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Receptor	Units	Ontario Hydro				Ministry of Natural Resources			
		2,4,5-T	TCDD			2,4,5-T	TCDD		
			Low[b]	Central	High		Low	Central	High
Recreational visitor (adult)	ug/kg/day	0.0[c]	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hunter/angler (total)	ug/kg/day	NA	0.0	0.0	0.0	NA	0.0	0.0	0.0

[a] *NA = not applicable.*
[b] *PHED = Pesticide Handler Exposure Database.*
[c] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[d] *The value 0.0 = <0.05 due to rounding.*

Table 6.21. *Estimates of resident margins of safety for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).*[a]

Receptor	Units	Ontario Hydro				Ministry of Transportation			
		2,4,5-T	TCDD			2,4,5-T	TCDD		
			Low[b]	Central	High		Low	Central	High
Resident (toddler)	µg/kg/day	0.0[c]	0.0	0.0	0.1	0.0	0.0	0.1	0.4
Hunter/angler child (toddler)	µg/kg/day		0.0	0.0	0.2		0.0	0.1	0.4

[a] *Blank cells = information not available.*
[b] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[c] *The value 0.0 = <0.05 due to rounding.*

Table 6.22. *Estimates of fish consumer margins of safety for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (µg/kg/day).*

	Ontario Hydro			Ministry of Natural Resources		
	Low[a]	Central	High	Low	Central	High
Angler-fish consumer	0.0[b]	0.0	0.0	0.0	0.0	0.0
First Nations-fish consumer	0.0	0.1	0.3	0.0	0.0	0.1

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[b] *The value 0.0 = <0.05 due to rounding.*

Table 6.23. *Estimates of wild game consumer margins of safety for 2,4,5-tetrachlorophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (µg/kg/day).*

Ontario Hydro	Moose			White-tailed deer		
	Low[a]	Central	High	Low	Central	High
Hunter-game consumer	0.0[b]	0.0	0.0	0.0	0.0	0.0
First Nations-game consumer	0.0	0.0	0.1	0.0	0.0	0.0
Ministry of Natural Resources	**Moose**			**White-tailed deer**		
	Low	Central	High	Low	Central	High
Hunter-game consumer	0.0	0.0	0.0	0.0	0.0	0.0
First Nations-game consumer	0.0	0.0	0.0	0.0	0.0	0.0

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[b] *The value 0.0 = <0.05 due to rounding.*

Table 6.24. *Estimates of bystander margins of safety for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)—1 day per year direct spray (Ontario Hydro).*

Year	Ground-based exposure at 30 m (µg/kg/day)				Aerial (fixed)-based exposure at 500 m (µg/kg/day)				Aerial (rotary)-based exposure at 500 m (µg/kg/day)			
	2,4,5-T	TCDD			2,4,5-T	TCDD			2,4,5-T	TCDD		
		Low[a]	Central	High		Low	Central	High		Low	Central	High
1948	0.2	0.0[b]	0.4	1.5[c]	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1949	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1950	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1951	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1952	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1953	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1954	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1955	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1956	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1957	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1958	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1959	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1960	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1961	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1962	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1963	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1964	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1965	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1966	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1967	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1968	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1969	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1970	0.2	0.0	0.4	1.5	0.1	0.0	0.1	0.7	0.1	0.0	0.2	0.8
1971	0.2	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0
1972	0.2	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0
1973	0.2	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0
1974	0.2	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0
1975	0.2	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0
1976	0.2	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0
1977	0.2	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0
1978	0.2	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0
1979	0.2	0.00	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.0

[a] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[b] *The value 0.0 = <0.05 due to rounding.*
[c] *Coloured font = exposure exceeded the TRV (TDI).*

Table 6.25. *Estimates of bystander margins of safety for 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)—1 day per year direct spray.*[a]

Year	Ground-based exposure at 30 m (µg/kg/day) Ministry of Transportation				Aerial (fixed)-based exposure at 500 m (µg/kg/day) Ministry of Natural Resources			
	2,4,5-T	TCDD			2,4,5-T	TCDD		
		Low[b]	Central	High		Low	Central	High
1953								
1954								
1955								
1956								
1957								
1958	0.1	0.0[c]	0.2	0.7	0.3	0.0	0.5	1.6[d]
1959	0.1	0.0	0.2	0.7	0.3	0.0	0.5	1.6
1960	0.1	0.0	0.2	0.7	0.3	0.0	0.5	1.6
1961	0.1	0.0	0.2	0.7	0.3	0.0	0.5	1.6
1962	0.1	0.0	0.2	0.7	0.3	0.0	0.5	1.6
1963	0.1	0.0	0.2	0.7	0.3	0.0	0.5	1.6
1964	0.1	0.0	0.2	0.7	0.3	0.0	0.5	1.6
1965	0.1	0.0	0.2	0.7	0.3	0.0	0.5	1.6
1966	0.1	0.0	0.2	0.7	0.3	0.0	0.5	1.6
1967	0.1	0.0	0.2	0.7	0.3	0.0	0.5	1.6
1968	0.1	0.0	0.2	0.7	0.3	0.0	0.5	1.6
1969	0.1	0.0	0.2	0.7	0.3	0.0	0.5	1.6
1970	0.1	0.0	0.2	0.7	0.3	0.0	0.5	1.6
1971	0.1	0.0	0.0	0.0	0.3	0.0	0.0	0.0
1972	0.1	0.0	0.0	0.0	0.3	0.0	0.0	0.0
1973	0.1	0.0	0.0	0.0	0.3	0.0	0.0	0.0

[a] *Blank cells = information not available.*
[b] *Low, central, and high exposure estimates are provided to bracket all potential levels of exposure between best (low) and worst (high) case scenarios; the central estimate is meant to capture the most realistic estimate.*
[c] *The value 0.0 = <0.05 due to rounding.*
[d] *Coloured font = exposure exceeded the TRV (TDI).*

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Appendix 2.1. Recruitment process for panel members

The following letter was sent to targeted professional associations on May 25, 2011, including the Canadian Institute of Forestry, Chemical Institute of Canada, International Society for Environmental Epidemiology, Ontario Professional Foresters Association, Society of American Foresters, Society of Toxicology of Canada, Inc., and Society of Toxicology.

The letter was also sent to several Ontario government ministries, including the Ministry of Citizenship and Immigration, Ministry of Education and Training, Ministry of Energy and Infrastructure, Ministry of the Environment, Ministry of Government Services, Ministry of Labour, Ministry of Municipal Affairs and Housing, Ministry of Northern Development and Mines, Ministry of Transportation, and Ontario Ministry of Agriculture Food and Rural Affairs for circulation to interested stakeholders and partners as appropriate.

Expression of Interest – Ontario Independent Fact Finding Panel on 2,4,5-T

The Ontario Independent Fact Finding Panel on 2,4,5-T (alone or as it was used in combination with other compounds) is seeking expressions of interest from suitably qualified scientists to serve on the Panel. Given the Terms of Reference of the Panel (see attachment); the Panel is particularly interested in hearing from individuals who have appropriate formal education, experience, and knowledge in the following areas:

1) Herbicide chemistry, with particular emphasis on the formation of polychlorinated contaminants

2) Exposure science, with particular emphasis on those involved directly or indirectly in pesticide application as well as those who may have been inadvertently exposed

3) Application methodology, with particular emphasis on herbicide application methodology in agricultural, forestry, industrial, including rights of way, and urban use scenarios

4) Epidemiology; priority consideration will be given to those with specific experience in the epidemiology of phenoxy herbicides

5) Toxicology; priority will be given to those with specific experience in human health outcomes related pesticide toxicology with emphasis on phenoxy herbicides

The Panel particularly encourages expressions of interest from individuals whose training and experience is of an interdisciplinary nature which includes any combination of the above skills. Panelists will be selected on the basis of formal education, experience, and recognition (which may include publication in the peer reviewed scientific literature, appointment to expert panels and invited lectures).

Panelists will be expected to commit to a part time undertaking over a period of 15-18 months from when the Panel is first convened. All Panelists will be required to satisfy strict Conflict of Interest guidelines. It is expected that the Panel members will function, largely, on a remote virtual basis, coming together as need dictates, at the discretion of the Chair. Limited travel within Ontario may also be required.

Interested individuals should submit a letter expressing their interest specifically highlighting the fields of expertise and qualifications noted above. All expressions of interest should be accompanied by a CV. References will be required.

Only electronic expressions of interest will be accepted and should be forwarded to the Chair, by June 10, 2011 to: Dr. Len Ritter at the following address Len.Ritter@245TPanelChair.ca. It is the intent to announce the composition of the Panel as soon as possible thereafter.

Leonard Ritter, PhD, Fellow Academy of Toxicological Sciences

Chair, Ontario Independent Fact Finding Panel on 2,4,5-T (alone or as it was used in combination with other compounds)

Ontario

BACKGROUNDER

Natural Resources

March 11, 2011

The Independent Fact-Finding Panel On Herbicide 2,4,5-T Terms Of Reference

BACKGROUND

2,4,5-Trichlorophenoxyacetic acid (2,4,5-T) herbicide was widely used in Ontario, dating back to the 1950s, 1960s, 1970s and possibly the 1980s. The Government of Ontario is concerned with the potential effect this herbicide may have on human health. 2,4,5-T is no longer registered for use in Canada.

The Ontario government takes the health and safety of Ontarians, including members of the Ontario Public Service past and present, very seriously. Therefore, the government is committed to fully understanding where, when and how 2,4,5-T was used in this province.

OBJECTIVE

The Independent Fact-Finding Panel on Herbicide 2,4,5-T will investigate its use by Government of Ontario ministries and agencies and examine whether exposure to 2,4,5-T herbicide may have potential health impacts.

The findings of the panel will be submitted to the Minister of Natural Resources and will be made available to the public.

MANDATE

The panel will, based on the information available:

- Investigate and document the scope and scale of the use of 2,4,5-T herbicide in the province by Ontario government ministries and agencies, including those acting as agents or as contractors;
- Determine the specific time period when 2,4,5-T herbicide was used in the province by Ontario government ministries and agencies;
- Determine the geographic area where 2,4,5-T herbicide was used in the province by Ontario government ministries and agencies;
- Examine whether exposure to 2,4,5-T herbicide in the affected areas may have potential health impacts;
- Document the methods 2,4,5-T herbicide was deployed by employees of provincial ministries and agencies, and the interaction of those employees and the general public with 2,4,5-T herbicide application operations in affected areas;
- Review the preparation, application and storage of 2,4,5-T herbicide as well as provincial occupational health and safety, and laws, standards and workplace practices including the use of personal protection equipment and applicable training in place at that time; and
- Refer, where appropriate, to the Workplace Safety and Insurance Board any findings that could assist its work.

MEMBERSHIP

There will be a full-time chair to manage, direct and oversee the day-to-day activities of the panel.

The chair will be supported by panel members who will serve on a part time, as needed basis. Panel members shall be selected based on the specific expertise required for the panel to execute its mandate.

2/...

The panel will be supported by a small team of support staff and be given the authority to appoint experts to complete research and studies.

REPORTING
The findings of the panel will be published in a report that will be submitted to the Minister of Natural Resources. The report of the panel will be released to the public.

The panel will work towards a final report by June 2012.

The panel may choose to develop interim reports and submit them to the Minister of Natural Resources. All interim reports will be released to the public.

The panel must also ensure that the privacy of individuals is protected as required by the Freedom of Information and Protection of Privacy Act.

In delivering its report(s) to the Minister, the panel shall be responsible for translation and printing and shall ensure the report is available simultaneously in both English and French, in electronic and printed formats, and in sufficient quantities for public release. The Ministry of Natural Resources shall be responsible for making the report available to the public.

PANEL ACTIVITIES
The panel shall perform its duties without expressing any conclusion or opinion about the liability of any person or organization and will not make any assessment regarding entitlement benefits.

In conducting its work, the panel shall follow Management Board of Cabinet directives and guidelines and other applicable government polices in obtaining services and goods considered necessary in their performance of their duties.

Within an approved budget, the panel chair may retain staff, investigators and expert advisers, as the chair considers necessary in the performance of the mandate at rates of remuneration approved by the Ministry of Natural Resources and will work with the Ministry of the Attorney General to obtain appropriate legal assistance where required. Persons retained shall be reimbursed for reasonable expenses incurred in connection with their duties in accordance with Management Board of Cabinet directives and guidelines.

ACCESS TO INFORMATION AND COOPERATION BY THE ONTARIO PUBLIC SERVICE
The panel chair may request any person to provide information or records to him, and hold public and or private meetings.

All ministries, Cabinet Office, the Premier's Office and all boards, agencies and commissions of the Government of Ontario shall, subject to any privilege or other legal restrictions, assist the panel to the fullest extent so that the mandate may be met.

The panel may request such information from the Ministry of Natural Resources and other government ministries it considers necessary to fulfill its mandate and, subject to any privilege or other legal restrictions, the Ministry of Natural Resources and other government ministries will cooperate with and assist the panel.

Appendix 3.1. Outcomes with inadequate or insufficient evidence

Outcomes for which the Institute of Medicine concluded evidence was inadequate or insufficient to determine association between exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) and adverse health effects

Cancers of the following:

- oral cavity, pharynx, and nasal cavity
- pleura, mediastinum, and other unspecified sites in the respiratory system and intrathoracic organs
- esophagus
- stomach
- colon/rectum
- hepatobiliary (liver and bile duct)
- pancreas
- bones, joints
- skin, breast reproductive organs
- urinary system, bladder kidney, brain/nervous system
- endocrine system
- other and unspecified sites
- blood (leukemia)

Other conditions:

- infertility
- spontaneous abortion (miscarriage)
- neonatal or infant death and stillbirth in offspring of exposed people
- low birth weight in offspring of exposed people
- birth defects in offspring of exposed people
- childhood cancer in offspring of exposed people
- neurodegenerative diseases, excluding Parkinson disease
- chronic peripheral nervous system disorders
- hearing loss
- respiratory disorders
- gastrointestinal, metabolic and digestive disorders
- immune system disorders
- circulatory disorders
- endometriosis
- effects on thyroid homeostasis
- eye problems
- bone conditions

Appendix 4.1. Ministry of Natural Resources herbicide spraying summary

Details for the spraying of approximately 161,143 acres of Ontario forests were available from review of all records provided to the panel that included herbicide usage (Ministry of Natural Resources database). The record references were reviewed to eliminate redundant descriptions of the same project. Information on district and townships were not always available, and some specific dates are also missing. The amounts and units used for arriving at the concentration and total sprayed varied from project to project. This table reproduces the given data. For purposes of exposure modelling, a value of 1.5 lbs per acre was used as a central value with high and low value extrapolations added.

District	Township	Year	Date	Modality[b]	Chemical(s)[c]	Amount used[d]	Acres treated	References[e]
[a]				G	2,4,5-T + 2,4-D	1 pint to 10 gals		A0137396
		1953		A	2,4,5-T	2,4, and 6 lbs/acre	30	A0130768, A0130853
		1956	Aug 20		2,4,5-T + 2,4-D	32 oz each a.e./gallon	0.5	A0133183
		1960	Aug 20	G	2,4,5-T + TCA	10 lbs TCA + 3 qts Brushkill in 200 gals water	8	A0130123
		1960	June	G	2,4,5-T	2 qts to 50 gals water		A0130528
		1964-1965			2,4,5-T			A0130529
		1971	Aug 4-6	A	2,4,5-T + 2,4-D	2 lbs a.e./acre; 3 gals/acre	1050	A0130794
		1976	July 29-Aug 2	G	2,4,5-T + 2,4-D	5 lbs/acre	30	A0130477
	Abinger	1978	June 19-July 14		2,4,5-T		280	A0129369
	Crerar	1961		G	2,4,5-T	2 qts Esteron to 10 gals fuel oil	160	A0133193
	Hebert	1973	May 28-June 8		2,4,5-T		265	A0127493
	Henry-Crerar	1962	May 16	G	2,4,5-T	76.6%; 2 oz to 4 oz/gal water; 35 gals used	600	A0128068
Boucher Lake	Clement	1956	Aug	A	2,4,5-T + 2,4-D and 2,4,5-T separately	2.5 lbs a.e; 3 gals/acre		A0130787
Brockville	Auga	1974	Sep 4	G	2,4,5-T + 2,4-D		20	A0129291
Cardiff	Cardiff	1965		G	2,4,5-T	2.3 oz a.e./gal; 3% by volume in diesel oil		A0128728
Chapleau								A0131211
Chapleau		1961	Aug	A	2,4,5-T	3 gals/acre	1660	A0133134, A0130806, A0133313
Chapleau	11H	1959	Aug 11-12	G	2,4,5-T	96 oz (1:10 solution)	30	A0133175
Chapleau	Panet	1958		A	2,4,5-T	96 oz a.e./gallon	325	A0133175
Cochrane	Case	1979		A	2,4,5-T + 2,4-D		100	A0133112
Cochrane	Colquhoun	1979		A	2,4,5-T + 2,4-D		659	A0133112
Cochrane	Fournier	1957	July 29-Aug 2	G	2,4,5-T + 2,4-D	.5 oz/gallon water	21	A0133168
Cochrane	Fournier	1957	July 29-Aug 2	G	2,4,5-T + 2,4-D	17 gals/acre; 356 gals and 178 oz	25	A0128723, A0128688, A0128690, A0128695

District	Township	Year	Date	Modality[b]	Chemical(s)[c]	Amount used[d]	Acres treated	References[e]
Cochrane	Fournier	1979		A	2,4,5-T + 2,4-D		109	A0133112
Cochrane	Leitch	1957	July 29-Aug 2	G	2,4,5-T + 2,4-D	1 pint/10 gals water	40	A0133168
Cochrane	Leitch	1958	Aug 4-19	A	2,4,5-T	4 gals/acre (3.7 gals water + 0.3 gals chemical)	1200	A0128686, A0133575, A0130787
Cochrane	Leitch	1979		A	2,4,5-T + 2,4-D		703	A0133112
Cochrane	Pyne	1957		A			800	A0128720, A0128719
Cochrane	Resume	1979		A	2,4,5-T + 2,4-D		198	A0133112
Espanola	Baldwin	1978	July 17-25		2,4,5-T + 2,4-D	4 lbs/acre	70	A0130499
Espanola	Deagle	1977	July 11-19	G	2,4,5-T + 2,4-D	5 lbs/acre	24	A0130510
Espanola	Deagle	1977	July 18-30	G	2,4,5-T + 2,4-D	2 gals 2,4-D; 1 gal 2,4,5-T, 40 gals fuel oil. 1 cup rhodamine dye	10	A0130478
Espanola	Deagle	1978	July 11-Aug 4	G	2,4,5-T + 2,4-D		120	A0130488
Espanola	Nairn	1976	July 28	G	2,4,5-T + 2,4-D	5 lbs/acre	42	A0130486
Espanola	Nairn	1976	July 22-24		2,4,5-T + 2,4-D	5 lbs a.i/acre (2,4-D amine and water: 2,4,5-T ester and fuel oil (10%); 3.75 lbs a.i./acre 2,4-D and 1.25 lbs a.i./acre 2,4,5-T)	53	A0130483, A0130484
Espanola	Nairn	1976	June 22-July 24		2,4,5-T + 2,4-D	5 lbs a.i/acre (2,4-D amine and water: 2,4,5-T ester and fuel oil (10%); 3.75 lbs a.i./acre 2,4-D and 1.25 lbs a.i./acre 2,4,5-T)	116	A0130482
Espanola	Nairn	1977	July 4-8	G	2,4,5-T + 2,4-D	4 lbs/acre	20	A0130505
Espanola	Tennyson	1978	June 21-26	G	2,4,5-T + 2,4-D	4 lbs/acre (fuel oil; 2,4-D 3 lbs/acre; 2,4,5-T 1 lb/acre)	20	A0130480
Espanola	Tennyson	1978	June 27-July 4		2,4,5-T + 2,4-D	4 lbs/acre	26	A0130490
Fort Frances		1963	Aug 14-30	G	2,4,5-T	76.8 oz a.e./gallon (3 pints to 45 gals)	116	A0133166
Fort Frances		1964	Aug 19-31	G	2,4,5-T	76.8 oz a.e./gallon (3 pints to 45 gals)	55	A0133167
Fort Frances	486924	1973	Aug	A	2,4,5-T + 2,4-D	12 total gals; 32 oz a.i.; 3 gals solution/acre (112% a.e.)	46	A0129516
Fort Frances	487922	1973	Aug	A	2,4,5-T + 2,4-D	56 total gals; 32 oz a.i.; 3 gals solution/acre (112% a.e.)	210	A0129505
Fort Frances	487924	1973	Aug 18-19	A	2,4,5-T + 2,4-D	22 total gals; 32 oz a.e.; 3 gals solution/acre (112% a.e.)	82	A0129513, A0129528, A0129529
Fort Frances	Carpenter	1953	Aug 31	G	2,4,5-T	5.4 kg/ha	19.5	A0129522
Fort Frances	Carpenter	1963	Aug 23-31	G	2,4,5-T	76.8 oz a.e./gallon at rate of 4 pints/100 gals water		A0129502, A0129521
Fort Frances	Carpenter	1963	Aug 23-31	G	2,4,5-T		37	A0129507
Fort Frances	Dance	1964	Aug 10-Sep 7	G	2,4,5-T	76.8 oz a.e./gallon (3 pints to 45 gals)	120	A0133165

District	Township	Year	Date	Modality[b]	Chemical(s)[c]	Amount used[d]	Acres treated	References[e]
Fort Frances	Dewart	1973	Aug	A	2,4,5-T + 2,4-D	78 total gals 32 oz a.i.; 3 gals solution/acre (112% a.e.)	300	A0129509
Fort Frances	Dewart	1973	Aug	A	2,4,5-T + 2,4-D	101 total gals 32 oz a.i.; 3 gals solution/acre (112% a.e.)	400	A0129511, A0129544, A0129545
Fort Frances	Farrington	1973	Aug	A	2,4,5-T + 2,4-D	134 total gals 32 oz a.i.; 3 gals solution/acre (112% a.e.)	522	A0129512, A0129533, A0129536, A0129540, A0129541, A0129543
Fort Frances	Kingsford	1963	Aug 19	G	2,4,5-T	5.4 kg/ha	37	A0129526, A0129502
Fort Frances	Kingsford	1963	Aug 20-31	G	2,4,5-T	76.8 oz a.e./gallon at rate of 4 pints/100 gals water		A0129502, A01295393
Fort Frances	Kingsford	1963	Aug 6-23	G	2,4,5-T	6.8 oz a.e./gallon at rate of 4 pints/100 gals water	100	A0129520
Fort Frances	Kingsford	1963	Sep 2-6	G	2,4,5-T	76.8 oz a.e./gallon (4 pints to 100 gal); 76 gals/acre	40	A0133186, A0133416
Fort Frances	Kingsford	1963	Sep 2-6	G	2,4,5-T	76.8 oz a.e./gallon at rate of 4 pints/100 gals water		A0129502, A0129527
Fort Frances	Kingsford	1963	Sep 2-16	G	2,4,5-T	76.8 oz a.e./gallon at rate of 4 pints/100 gals water	40	A0129521
Fort Frances	McCrosson	1968	Aug 20-Sep 3	G	2,4,5-T	76.8 oz a.e./gallon 2,4,5-T mixed at rate of 2.4 gals/100 gals water	130	A0129517, A0131175
Fort Frances	Morson	1963	Aug 6-29	G	2,4,5-T	76.8 oz a.e./gallon at rate of 4 pints/100 gals water	160	A0129506, A0129545
Fort Frances	Rowe	1968		A	2,4,5-T + 2,4-D	76.8 oz a.e./gallon at rate of 4 pints/100 gals water	2690	A0129508, A0131175
Fort Frances	Rowe	1973	Aug 31	A	2,4,5-T	2.2 (no units)	296	A0129524
Fort Frances	Rowe	1973	Aug	A	2,4,5-T + 2,4-D	157 total gals 32 oz a.i.; 3 gals solution/acre (112% a.e.)	620	A0129510, A0129542
Geraldton		1979		A	2,4,5-T + 2,4-D		305	A0133112
Geraldton		1979		A	2,4,5-T + 2,4-D		1130	A0133112
Geraldton		1979		A	2,4,5-T + 2,4-D		705	A0133112
Geraldton		1979		A	2,4,5-T + 2,4-D		978	A0133112
Geraldton	491881	1969	July 24-Aug 7	A	2,4,5-T + 2,4-D	3 gals/acre	964	A0130835, A0130836, A0130837, A0130844, A0131251
Geraldton	493871	1967	Aug 14-17	G	2,4,5-T + 2,4-D	3 gals/acre	10	A0131253
Geraldton	495863	1964	July 8-9	A	2,4,5-T + 2,4-D and 2,4,5-T separately	3.25 gals/acre	252	A0131254
Geraldton	495863	1967	July 25-29	A	2,4,5-T + 2,4-D	3 gals/acre	1079	A0131255
Geraldton	497861	1963	Summer	A	2,4,5-T	1.25 lbs a.e./acre in 3.3 gals of water/acre	374	A0131257

District	Township	Year	Date	Modality[b]	Chemical(s)[c]	Amount used[d]	Acres treated	References[e]
Geraldton	497862	1963	July	A	2,4,5-T	1.25 lbs a.e./acre in 3.3 gals of water/acre	884	A0131259
Geraldton	497862	1967	July 25-29	A	2,4,5-T	1.25 lbs a.e./acre in 3 gals of water/acre	942	A0131260
Geraldton	497862, 497861	1969	July 22-24	A	2,4,5-T + 2,4-D	1.2 lbs a.e./acre; 3 gals/acre	2010	A0131261, A0131258
Geraldton	498861	1965	July 11-12		2,4,5-T + 2,4-D	1.25 lbs a.e./acre; 3.25 gals/acre	1383	A0131262
Geraldton	498862	1965	July 11-12	G	2,4,5-T + 2,4-D	1.25 lbs a.e./acre; 3 gals/acre	948	A0131264
Geraldton	498862	1967	July 25-29	A	2,4,5-T + 2,4-D	1.25 lbs a.e./acre; 3 gals/acre		A0131265
Geraldton	503871	1967	July 30-Aug 1		2,4,5-T	3 gals/acre	500	A0130833, A0132736
Geraldton	BM 493852	1969	July 18-21	A	2,4,5-T + 2,4-D	1.3 lbs a.e./acre; 3 gals/acre	1670	A0129498
Geraldton	BM 493854	1969	July 18	A	2,4,5-T + 2,4-D	1 lb a.e./acre	909	A0129499, A0129498
Geraldton	BM 494853	1969	July 19-21	A	2,4,5-T + 2,4-D	1.3 lbs a.e./acre; 3 gals/acre	756	A0129496, A0129498
Geraldton	BM 495853	1969	July 22-Aug 6	A	2,4,5-T + 2,4-D	1.3 lbs a.e./acre; 3 gals/acre	460	A0129495, A0129498
Geraldton	BM 495854	1969	July 22	A	2,4,5-T + 2,4-D	1.3 lbs a.e./acre	337	A01295, A0129498
Geraldton	BM498861	1969	July 19-21	G	2,4,5-T + 2,4-D	1.2 lbs a.e./acre; 3 gals/acre	3410	A0131263
Gillies	Eldridge	1969	July 21-25	A			1000	A0127359
Gillies	Eldridge	1969	May 12-June 13	G			348	A0127365
Gillies	Eldridge	1971	May 20-28	G	2,4,5-T	1:100 (112 oz); 816 oz of chemical (5 gals + 16 oz)	68	A0127430
Gogama	Burrows	1961	Aug 20-21	A	2,4,5-T	96 oz a.e./gal; 28.8 oz a.e./acre	1200	A0131179, A0131206
Gogama	Burrows, Cabot	1964	July 25-Aug 9	A	2,4,5-T	3 gals/acre	1400	A0133640
Gogama	Kemp, Burrows	1963	Aug	A	2,4,5-T	3 gals/acre	900	A0133639
Gogama	Kemp, Burrows, Cabot	1965	July 27-Aug 5	A	2,4,5-T	3 gals/acre	920	A0133641
Gore	Charlottenburgh	1970			2,4,5-T		80	A0129262
Haldimande, Cramahe		1970		A	2,4,5-T + 2,4-D			A0130763
Hastings	Marmora	1977					2	A0130533
Hearst	238	1979		A	2,4,5-T + 2,4-D		1048	A0133112
Hearst	289	1979		A	2,4,5-T + 2,4-D		550	A0133112
Hearst	Bannerman	1979		A	2,4,5-T + 2,4-D		524	A0133112
Hearst	Fushimi	1979		A	2,4,5-T + 2,4-D		1386	A0133112
Huronia	Vespra	1979		G	2,4,5-T			A0129565
Kapuskasing		1952	Nov 11-12	G	2,4,5-T		1.5	A0131231, A0131281
Kapuskasing		1953	July 28	G	2,4,5-T		1	A0131230
Kapuskasing		1956	July 5-10	A	2,4,5-T	1 lb a.e; 3.3 gals/acre	600	A0131286, A0131278, A0131233
Kapuskasing		1957	July 11-16	A	2,4,5-T		500	A0131233

District	Township	Year	Date	Modality[b]	Chemical(s)[c]	Amount used[d]	Acres treated	References[e]
Kapuskasing		1958	July 16-23	A			1456	A0131234, A0169135
Kapuskasing		1958	July 7-10	A	2,4,5-T		636	A0131235
Kapuskasing		1966	July 19-21	A	2,4,5-T + 2,4-D	4 gals/acre, 1.5 lbs a.e./ acre, and 1.8 lbs a.e./acre, respectively	716	A0128594, A0131192, A0130156
Kapuskasing		1969	July 16-19	A	not specified	2 lbs a.e./acre; 3 gals/acre	302	A0128595, A0131192
Kapuskasing	238	1968		A	2,4,5-T + 2,4-D	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre (2,4-D - 2,4,5-T mix)	2222	A0130158
Kapuskasing	238	1969		A	2,4,5-T + 2,4-D	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre (2,4-D - 2,4,5-T mix)		A0130159
Kapuskasing	238	1969		A	2,4,5-T + 2,4-D	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre (2,4-D - 2,4,5-T mix)		A0130158
Kapuskasing	Abbott	1979		A	2,4,5-T + 2,4-D		168	A0133112
Kapuskasing	Bourinot	1960	July 3-9	A	2,4,5-T iso-octyl ester	1.5 lbs a.e./acre; 3.33 gals/acre	460	A0169140
Kapuskasing	Bourinot (234)	1966	July 5-7	A	2,4,5-T + 2,4-D	810 gals; 48 oz/gal a.e.; 2 lbs a.e./acre	702	A0128443, A0131192
Kapuskasing	Carmichael, MacVicar	1959	May 28-June 6; July 13-17	A	2,4,5-T	1.5 lbs a.e./acre; 3.33 gals/acre	1425	A0128627, A0169137
Kapuskasing	Casselman	1978	July 21	A	2,4,5-T + 2,4-D	2 lbs acid/acre; 3 gals/acre; 96 oz/gallon	70	A0128448
Kapuskasing	Casselman	1979		A	2,4,5-T + 2,4-D		1988	A0133112
Kapuskasing	Clay	1978	July 17-18	A	2,4,5-T + 2,4-D	2 lbs acid/acre; 3 gals/acre; 96 oz/gallon	750	A0128476
Kapuskasing	Cumming	1977	July 24-Aug 2	A	2,4,5-T + 2,4-D	2 lbs acid/acre; 3 gals/acre	2040	A0128479
Kapuskasing	Cumming, Torrance, Gurney, Deetzel	1964	June 26-July 6	A	2,4,5-T + 2,4-D and 2,4,5-T separately	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre (2,4-D + 2,4,5-T mix)	3081	A0130153, A0131192, A0130156, A0128578, A0133191, A0133474
Kapuskasing	Cumming	1964	June 26-28	A	(a) 2,4,5-T iso octyl ester (b) 2,4,5-T + 2,4-D (c) 2,4,5-T + 2,4-D (d) 2,4,5-T + 2,4-D	(a) 96 oz at 1.5 lbs a.e./acre (b) 32 oz each at 2 lbs a.e./ acre (c) 32 oz each at 2 lbs a.e./ acre (d) 38.4 oz each at 2 lbs a.e./ acre; 3.25 gals of mixture/acre for all	40	A0128483, A0131192
Kapuskasing	Fauquier	1968		A	2,4,5-T + 2,4-D	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre (2,4-D + 2,4,5-T mix)	688	A0130158
Kapuskasing	Fauquier	1969		A	2,4,5-T + 2,4-D	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre (2,4-D + 2,4,5-T mix)		A0130159

District	Township	Year	Date	Modality*	Chemical(s)*	Amount used*	Acres treated	References*
Kapuskasing	Fergus	1968		A	2,4,5-T + 2,4-D	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre (2,4-D + 2,4,5-T mix)	1368	A0130158
Kapuskasing	Fergus	1968	July 3-12	A	2,4,5-T + 2,4-D	2 lbs acid/acre; 3 gals/acre	1368	A0128486, A0128527; A0169154
Kapuskasing	Fergus	1979		A	2,4,5-T + 2,4-D		132	A0133112
Kapuskasing	Fergus, Opasatika, Ecclestone	1962	July 15-20	A	2,4,5-T iso-octyl	1.5 lbs a.e./acre; 3.33 gals/ acre	1973	A0169143
Kapuskasing	Fintry	1959	July 21-27	A	2,4,5-T	4.86 gals; 0.33 lbs a.e./gal; 1.6 lbs a.e./acre	350	A0128409, A0128410, A013314, A0133355, A0131192
Kapuskasing	Griffen	1979		A	2,4,5-T + 2,4-D		720	A0133112
Kapuskasing	Guilfoyle	1965	July 13	A	2,4,5-T + 2,4-D	4 lbs a.e./acre		A0128489
Kapuskasing	Guilfoyle	1965	June 26-July 8	A	2,4,5-T + 2,4-D	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre 2,4,D + 2,4,5-T mix)	3017	A0128489
Kapuskasing	Gurney	1964	June 30-July 5	A	2,4,5-T iso-octyl ester 2,4,5-T + 2,4-D 2,4,5-T + 2,4-D 2,4,5-T + 2,4-D	3.25 gal/acre all segments (a) 96 oz, 1.5 lbs a.e./acre (b) 32 oz each at 2 lbs a.e./ acre (c) 32 oz each at 2 lbs a.e./acre (d) 38.4 oz each at 2 lbs a.e./ acre	1270	A0128493, A0131192
Kapuskasing	Hopkins	1965	June 26-27	A	2,4,5-T iso-octyl ester 2,4,5-T + 2,4-D 2,4,5-T + 2,4-D	3.25 gal/acre all segments (a) 96 oz at 1.5 lbs a.e./acre (b) 96 oz at 1.5 lbs a.e./acre (c) 38.4 oz each at 2 lbs a.e./ acre	3017	A0128499, A0128648, A0131192, A0130154
Kapuskasing	Hopkins	1978	July 18-21	A	2,4,5-T + 2,4-D	3 gals/acre; 2 lbs a.e./acre	1800	A0128496
Kapuskasing	Idlington	1966		A	2,4,5-T		250	A0128522
Kapuskasing	Kendall	1957	Aug 21-25	A	2,4,5-T	1.5 lbs acid/acre; 4 gals/acre	600	A0128537, A0133349, A0133144; A0131213
Kapuskasing	Lisgar, Seaton, Fenton	1967	July 16	A	2,4,5-T + 2,4-D	1.8 lbs acid/acre; 3 gals/acre	110	A0128485
Kapuskasing	Magladery	1967	July 19-20	A	2,4,5-T + 2,4-D	1.8 lbs acid/acre; 3 gals/acre	1986	A0128485, A0128521
Kapuskasing	Magladery	1968	July 5-6	A	2,4,5-T + 2,4-D	2 lbs acid/acre; 3 gals/acre	1328	A0128486, A0128527, A0169154
Kapuskasing	Maude	1960	July 3-9	A	2,4,5-T iso-octyl ester	1.5 lbs a.e./acre; 3.33 gals/ acre	705	A0169140
Kapuskasing	McCowan	1979		A	2,4,5-T + 2,4-D		825	A0133112

District	Township	Year	Date	Modality[b]	Chemical(s)[c]	Amount used[d]	Acres treated	References[e]
Kapuskasing	McCrea	1969		A	2,4,5-T + 2,4-D	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre (2,4D + 2,4,5T mix)		A0130159
Kapuskasing	McCrea	1978	July 24-25	A	2,4,5-T + 2,4-D		150	A0128505
Kapuskasing	McMillan	1959	July 21-27	A	2,4,5-T	4.86 gals; 0.33 lbs a.e./gallon; 1.6 lbs a.e./acre	650	A0128410, A0133355, A0131192, A0133144
Kapuskasing	McMillan	1961	July 11-16	A	2,4,5-T	1.5 a.e./acre	200	A0128411, A0131192, A0133361
Kapuskasing	McVicar	1979		A	2,4,5-T + 2,4-D		200	A0133112
Kapuskasing	Nansen	1961	July 11-16	A	2,4,5-T	1.5 lbs a.e./acre	150	A0128550, A0133361, A0131192
Kapuskasing	Nansen	1963	Aug 7-9	A	2,4,5-T	1.5 lbs a.e./acre	445	A0128577, A0131168, A0133463, A0131192
Kapuskasing	Nansen	1979		A	2,4,5-T + 2,4-D		2329	A0133112
Kapuskasing	O'Brien	1966	July 4-14	A	2,4,5-T + 2,4-D	3 gals/acre	2089	A0128507, A0128522
Kapuskasing	Opasatika	1968	July 10-12	A	2,4,5-T + 2,4-D	2 lbs acid/acre; 3 gals/acre	1240	A0128486, A0128527, A0169154
Kapuskasing	Opasatika	1978	July 22	A	2,4,5-T + 2,4-D	3 gals/acre	1130	A0128508
Kapuskasing	Parnell	1964	June 26-July 6	A	2,4,5-T	96 oz at 1.5 lbs a.e./acre	1169	A0128518, A0131192
Kapuskasing	Parnell	1977	Jul 24-Aug 2	A	2,4,5-T + 2,4-D	2 lbs acid/acre; 3 gals/acre	220	A0128514
Kapuskasing	Ritchie	1968		A	2,4,5-T + 2,4-D	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre (2,4-D + 2,4,5-T mix)	1074	A0130158
Kapuskasing	Rogers	1962	July 16-19	A	2,4,5-T	1.5 lbs a.e./acre	437	A0128593, A0131192
Kapuskasing	Rogers	1962	July 16-19	A	2,4,5-T	1.5 lbs a.e./acre	669	A0128553, A0128597, A0131192
Kapuskasing	Rogers	1964	July 9-14	A	2,4,5-T	1.5 lbs a.e./acre	796	A0128578, A0131192, A0130153, A0133191
Kapuskasing	Rogers	1965	July 14-15	A	2,4,5-T	1.5 lbs a.e./acre	578	A0128599, A0131192, A0128583, A0130154
Kapuskasing	Rogers	1966	July 19-21	A	2,4,5-T + 2,4-D	4 gals/acre; 1.5 lbs a.e./ acre and 1.8 lbs a.e./acre, respectively	403	A0128408, A0128592, A0131192, A0130156

District	Township	Year	Date	Modality[a]	Chemical(s)[c]	Amount used[d]	Acres treated	References[e]
Kapuskasing	Rogers	1968		A	2,4,5-T + 2,4-D	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre (2,4-D + 2,4,5-T mix)	1072	A0130158
Kapuskasing	Seaton	1961	July 6-11	A	2,4,5-T iso-octyl	1.5 lbs a.e./acre; 3.33 gals/ acre	1585	A0169141
Kapuskasing	Seaton	1967	July 5-6	A	2,4,5-T + 2,4-D	1.8 lbs a.e./acre (339 gals)	2555	A0128521, A0131192, A0128485, A0169153
Kapuskasing	Shackleton	1979		A	2,4,5-T + 2,4-D		535	A0133112
Kapuskasing	Shanley	1966	July 4-8	A	2,4,5-T + 2,4-D	2 lbs acid/acre; 3 gals/acre	884	A0128522, A0128443, A0128507
Kapuskasing	Shearer	1964	June 26-July 6	A	2,4,5-T	(a) 450 gals (b) 90 gals	960	A0128524
Kapuskasing	Shearer	1968	Jul 3-7	A	2,4,5-T + 2,4-D	2 lbs acid/acre; 3 gals/acre	2624	A0128486, A0128527, A0169154
Kapuskasing	Shearer	1979		A	2,4,5-T + 2,4-D		2112	A0133112
Kapuskasing	Studholme	1961	July 11-16	A	2,4,5-T	1.5 lbs a.e./acre	650	A0128542, A0131192, A0133361
Kapuskasing	Studholme	1968		A	2,4,5-T + 2,4-D	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre (2,4-D + 2,4,5-T mix)	834	A0130158
Kapuskasing	Studholme	1969		A	2,4,5-T + 2,4-D	1.5 lbs a.e./acre (2,4,5-T) and 2 lbs a.e./acre (2,4-D + 2,4,5-T mix)		A0130159
Kapuskasing	Teetzel	1964	June 30 and July 4	A	2,4,5-T	1.5 lbs a.e./acre; 3.5 gals/acre		A0128528, A0131192, A0128493
Kemptville	Bathurst	1966	Feb	G	2,4,5-T	4 oz/gallon	16	A0129366
Kemptville	Crown	1967-1968	Apr-Mar	A			7	A0130151
Kemptville	Dalhousie	1968	Sep 4-Feb 4	G	2,4,5-T		210	A0129367
Kemptville	Darling	1969	Feb 5-Mar 29	G	2,4,5-T		145	A0129368
Kemptville	Lanark Forest	1969-1970	Apr-Mar	A	2,4,5-T		44	A0130155
Kemptville	Larose	1966-1967	Apr-Mar	A			210	A0130150
Kemptville	Larose	1967-1968	Apr-Mar	A			195	A0130151
Kemptville	Limerick Forest	1968-1969	Apr-Mar	A				A0130152
Kemptville	NCC Greenbelt Forest	1968-1969	Apr-Mar	A				A0130152
Kemptville	Williamsburgh Township Forest	1966-1967	Apr-Mar	A			22	A0130150
Kemptville	Williamsburgh Township Forest	1967-1968	Apr-Mar	A			12	A0130151
Kenora		1969	May 5-June1	G	2,4,5-T		428	A0130607, A0130608
Kenora		1974	Aug 19-28	A	2,4,5-T + 2,4-D	3 lbs a.e./acre; 6 gals/acre	434	A0130603
Kenora	Kenora Forest	1962	Aug 6-15	G	2,4,5-T	2.5 lbs/acre	528	A0130604, A0133145, A0133360

District	Township	Year	Date	Modality[b]	Chemical(s)[c]	Amount used[d]	Acres treated	References[e]
Kirkland Lake	Bownan	1979		A	2,4,5-T + 2,4-D		30	A0133112
Kirkland Lake	Doon	1979		A	2,4,5-T + 2,4-D		200	A0133112
Kirkland Lake	Tolstoi	1979		A	2,4,5-T + 2,4-D		70	A0133112
Kirkland Lake	Willet	1979		A	2,4,5-T + 2,4-D		350	A0133112
Lake Huron		1961-1962	Apr-Mar	A	2,4,5-T		45	A0129585
Lake Huron		1962-1963	Apr-Mar	A	2,4,5-T		32	A0129589
Lake Huron		1964-1965	Apr-Mar	G	2,4,5-T		12	A0133128
Lake Huron		1964-1965	Apr-Mar	G	2,4,5-T		20	A0133128
Lake Huron		1965-1966	Apr-Mar	A	2,4,5-T		48	A0129590
Lake Huron	Albermarle	1969	Feb		2,4,5-T			A0129567
Lake Huron	Collingwood	1963	Apr-Mar	G	2,4,5-T		106	A0133124
Lake Huron	Collingwood	1967-1968	Apr-Mar	A	2,4,5-T		30	A0129591
Lake Huron	Collingwood	1968	Dec-Jan 1969	G	2,4,5-T		50	A0129577
Lake Huron	Collingwood	1968	Jan	G	2,4,5-T		30	A0129581
Lake Huron	Collingwood	1969	Dec	G	2,4,5-T		33	A0129576
Lake Huron	Collingwood	1969	Dec	G	2,4,5-T		29	A0129573
Lake Huron	Collingwood	1969-1970	Apr-Mar	A	2,4,5-T			A0129584
Lake Huron	Collingwood, Euphrasia, Holland							A0129566, A0132773
Lake Huron	Euphrasia	1963	Apr-Mar	G	2,4,5-T		50	A0133124
Lake Huron	Euphrasia	1963	Apr-Mar	G	2,4,5-T		12	A0133125
Lake Huron	Glenelg	1963	Apr-Mar	G	2,4,5-T		11	A0133124
Lake Huron	Holland	1963	Apr-Mar	G	2,4,5-T		18	A0133125
Lake Huron	Holland	1965-1966	Apr-Mar	A	2,4,5-T		78	A0129571
Lake Huron	Keppel	1964-1965	Apr-Mar	A	2,4,5-T		60	A0129586
Lake Huron	Keppel	1969	Feb 3-Mar 6		2,4,5-T		100	A0129570
Lake Huron	Keppel, Albermarle	1968-1969	Apr-Mar	A	2,4,5-T			A0129588
Lake Huron	Keppel, Sullivan	1971-1972	Apr-Mar	A	2,4,5-T		80	A0129587
Lake Huron	Nicholl	1968			2,4,5-T			A0130531
Lake Huron	Osprey	1967-1968	Apr-Mar	A	2,4,5-T		30	A0129591
Lake Huron	Puslinch	1965		G	2,4,5-T		11	A0130527
Lake Huron	Sydenham	1963	Apr-Mar	G	2,4,5-T		10	A0133124
Lake Huron	Sydenham	1963	Apr-Mar	G	2,4,5-T		46	A0133125
Lake Huron	Sydenham	1966-67	Apr-Mar	G	2,4,5-T		48	A0133127
Lake Huron	West Luther	1968		G	2,4,5-T		10	A0130532
Leeds County	Kitley	1977	Sep 9-13	G	2,4,5-T + 2,4-D			A0129286
Longlac		1952	Aug 18	G	2,4,5-T		1	A0131311
Longlac		1952	Aug 13-14	G	2,4,5-T		3	A0131310
Longlac		1954	July 23		2,4,5-T + 2,4-D		0.5	A0131313
Longlac		1954	July 21-22	G	2,4,5-T		3	A0131312
Longlac		1955	July		2,4,5-T			A0131317, A0131316, A0131301
Longlac		1955	July		2,4,5-T			A0131316

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Longlac		1955	June	G	2,4,5-T			A0131314
Longlac		1957	Jan	A	2,4,5-T	1 lb a.e./acre; in 3.3 gals water/acre	432	A0131318
Longlac		1958	July 22-Aug 3	G	2,4,5-T	3 lbs-7 lbs a.e.; 4.5 gals/acre		A0131289
Longlac		1959	July 5-10	A	2,4,5-T	1.25 lbs a.e.; 3.3 gals/acre	964	A0131290
Longlac		1960		A	2,4,5-T		206	A0131299
Longlac		1960	July 1-12	A	2,4,5-T	1.25 lbs a.e.; 3.25 gals/acre	1481	A0131291, A0131300, A0131305
Longlac		1961	June 21-Aug 5	G	2,4,5-T	6 lbs a.e.; 4.0 gals/acre	548	A0131292, A0131296, A0131297
Longlac		1963	June 26-27	A	2,4,5-T	6 lbs a.e./3 gals/acre	884	A0131269
Longlac		1964	July 8-9	A	2,4,5-T + 2,4-D and 2,4,5-T separately	6 lbs a.e./gallon; 3.25 gals/ acre	665	A0131267
Longlac		1965	July 11-12	A	2,4,5-T + 2,4-D and 2,4,5-T separately	6 lbs a.e./gallon; 3.25 gals/ acre	2415	A0131272, A0131274, A0131295
Longlac		1967	July 25-29	A	2,4,5-T + 2,4-D	1.25 lbs a.e./acre; 3 gals/acre	564	A0131268, A0131271, **A0131287**
Longlac	BM498861	1969	July 18-21		2,4,5-T + 2,4-D and 2,4,5-T separately	1.25 lbs a.e./acre; 3 gals/acre	3410	A0131273, A0131297
Marten River	McLaren, Gladman	1968	May 21-June 3	G	2,4,5-T	10 gals	252	A0127573
North Bay	Bastedo	1960	May 11-23	A	2,4,5-T	2.5 gals	170	A0128074
North Bay	Bastedo	1964	May 28-June 25	G	2,4,5-T	112 a.e.; 1.5 oz/gallon water but slightly increased at end of project; 18 gals; 20 gals/acre	360	A0128072, A0128071
North Bay	Boulter	1966	July 22-Aug 6	G	2,4,5-T + 2,4-D		54	A0127302
North Bay	Boulter	1968	Aug 12-16	G	2,4,5-T + 2,4-D	22.4 l/ha (liquid rate) [14.9 gals/acre]	16	A0127301
North Bay	Clement	1956	Aug 13-19	A	2,4,5-T + 2,4-D	.5 gal emulsion 2.5 gals water; 3 gals/acre	274	A0133159, A0133373, A0133174
North Bay	Falconer	1966	May 27-June 9	G	2,4,5-T	1.54 to 2 oz/gallon	296	A0127765, A0127767
North Bay	McLaren	1957	May 22-June 23	G	2,4,5-T	20 gals total chemicals: 112 a.e.; 1.5 oz/gallon in small sprayer and 4 oz/gallon solo mister	422	A0127876
North Bay	McLaren	1958	July 22	A	2,4,5-T	4 gals to 36 gals water [1221 gals herbicide used]; 3 gals/ acre	4068	A0127797, A0127805
North Bay	McLaren	1963	May-June	G	2,4,5-T	1.5 oz/gallon water	544	A0128026
North Bay	McLaren	1964	May 19-June 30	G	2,4,5-T	112 a.e.; 1.5 oz/gallon water; 25 gals used	428	A0128027
North Bay	McLaren	1966	May 30-June 17	G	2,4,5-T	25 gals	288	A0127770
North Bay	McLaren	1966	May 31-June 15	G	2,4,5-T	20 gals	300	A0127530
North Bay	Sheppard	1956		A	2,4,5-T		274	A0130785
North Bay	Vogt, Phyllis, Belfast, Joan	1957	Aug 15-22	A	2,4,5-T	74.6% a.e. (1.5 qts/3 gals)	3000	A0133159

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Ottawa		1979		A	2,4,5-T		57	A0133112
Ottawa	Fitzroy	1978	Aug 17-31	G	2,4,5-T + 2,4-D	59.4 gals/acre; 50 oz a.i./acre in water		A0129234
Ottawa	Gloucester	1979		A	2,4,5-T		63	A0133112
Ottawa	Huntley Township	1973	July 7-9	G	2,4,5-T + 2,4-D	3.1 lbs a.e./acre; 24 gals/acre	23	A0129257
Ottawa	Osgoode	1978	Jul 6	G	2,4,5-T + 2,4-D	24 gals/acre; 50 oz a.i./acre in water		A0129250
Parry Sound	Ballantyne	1959	Aug 17-28	G	2,4,5-T	4 oz/gallon	128	A0133162
Parry Sound	Conger	1954	Nov	A	2,4,5-T	76.8 oz/gallon a.e.		A0130854, A0130785
Parry Sound	Franklin	1961	June 13-23	G	2,4,5-T	2.4 oz/gallon solution (2% by volume)	30	A0133162
Parry Sound	Livingston	1959	Aug 3-14	G	2,4,5-T	2.4 oz/gallon solution (2% by volume)	21	A0133162, A0133187, A0133381
Pembroke	Brougham	1975	July 22-25	G	2,4,5-T		25	A0130597
Pembroke	Head	1969	Aug 3	A	2,4,5-T	3 lbs/acre	420	A0130599
Pembroke	Head	1970	Aug 10	A	2,4,5-T			A0130600
Pembroke	Murchinson	1968	May 14-June 14	G	2,4,5-T	1.8 lbs a.e./acre	244	A0129339
Pembroke	Murchinson	1969	Aug 5-6	G	2,4,5-T	1.5 lbs a.e./acre	200	A0129353
Pembroke	Murchinson	1969	June 3-25	G	2,4,5-T		136	A0129338
Pembroke	Murchinson	1970	May 20-June 25	G	2,4,5-T		180	A0129337
Port Arthur	Black Sturgeon	1956	late Aug	G	2,4,5-T + 2,4-D	1.2 lbs/acre and 0.6 lbs/acre		A0133414
Port Arthur	Dog River Concession	1956	June 28; Aug 8 and late Aug	G	2,4,5-T + 2,4-D	1 lb/acre		A0133414
Port Arthur	Fraleigh	1969	May 8	A	2,4,5-T + 2,4-D	180 gals/trip	576	A0130796
Port Arthur	Goldie	1969		G	2,4,5-T + 2,4-D	[16.5 gals herbicide containing 7 lbs/gallon herbicide in 164 gals water]	15	A0130797
Port Arthur	McIntyre	1960	July 28-Aug 1	G	2,4,5-T	1.2 lbs/acre	10	A0133164, A0133415
Port Arthur	McIntyre	1960	July 28-Aug 1	G	2,4,5-T			A0130792, A0130791
Port Arthur	Nipigon Concession	1956	July 7-9	A	2,4,5-T + 2,4-D	1.2 lbs/acre	450	A0133414
Red Lake		1979		A	2,4,5-T + 2,4-D		480	A0133112
Sudbury	Carlysle	1968			2,4,5-T + 2,4-D	"mixture of 30 l"	275	A0130497
Sudbury	Struthers	1976	Apr 25-May 12		2,4,5-T + 2,4-D		337	A0130503
Sudbury	Struthers	1978	June 6-30	G	2,4,5-T + 2,4-D		90	A0130504
Sudbury	Waldie	1976		G	2,4,5-T + 2,4-D		103	A0130502
Swastika	Cairo	1965	July 30	A	2,4,5-T	1.9 lbs/acre	292	A0130521
Swastika	Dunmore	1963	Aug	A	2,4,5-T	1.9 lbs/acre	96	A0130520
Swastika	Dunmore	1963	Aug	A	2,4,5-T	1.9 lbs/acre	206	A0130519
Swastika	Gross	1962	Aug 14-18	A	2,4,5-T	1.9 lbs/acre; 96 oz/gal a.e.	520	A0130522, A0131170, A0133418, A0133189, A0133191

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Swastika	Gross	1963	Aug	A	2,4,5-T	1.9 lbs/acre	252	A0130516
Swastika	Holmes	1963	Aug	A	2,4,5-T	1.9 lbs/acre	140	A0130518
Swastika	Truax	1962	Aug 18	A	2,4,5-T	used double concentration since sprayed twice; 1.9 lbs/ acre for each application	11	A0130515
Swastika	Burt	1963	Aug	A	2,4,5-T	1.9 lbs/acre	88	A0130517
Temagami	Belfast	1963	July 15- Aug 15			10% solution; 3 gals/acre	660	A0127429
Temagami	Belfast	1972	June 12-July 7	G	2,4,5-T	10 gals	194	A0127492, A0127491
Temagami	Eldridge	1972	May 15-29	G	2,4,5-T	10 gals	166	A0127490, A0127489
Temagami	Joan	1964	July	A	2,4,5-T	1.5 lbs a.e./acre; 3 gals/acre		A0127170, A0127487, A0127342, A0127487, A01273425, A0127461
Temagami	Joan	1971	May 17-21	G	2,4,5-T	10 gals chemical, 2,4,5-T (112 oz)	180	A0127431
Thunder Bay		1965	Aug 5-20	A	2,4,5-T + 2,4-D	4 gals/acre	6233	A0130801, A0130800
Thunder Bay	488884	1977	July 24-26/July 26-27	A	2,4,5-T + 2,4-D	2.5 lbs acid equivalent; 3 gals/acre	1050	A0130799
Thunder Bay	488884	1977	July 24-26/July 26-27	A	2,4,5-T + 2,4-D	2 lbs acid equivalent; 3 gals/ acre	456	A0130799
Thunder Bay	491853	1978	Aug 27- 29	G	2,4,5-T	2.6 lbs a.e./acre	18	A0129487, A0129489
Thunder Bay	493653	1977	July 26-28	G	2,4,5-T	2.1 lbs a.e./acre	13	A0129492, A0133161
Thunder Bay	493852	1977	July 26-28	G	2,4,5-T	2.1 lbs a.e./acre	31	A0129493
Thunder Bay	493853	1978	July 29- Aug 16	G	2,4,5-T	2.6 lbs a.e./acre	43	A0129485, A0129489, A0129492, A0129493
Thunder Bay	493854	1978	Aug 17-25	G	2,4,5-T	2.6 lbs a.e./acre	29	A0129486, A0129489, A0129488
Thunder Bay	493892	1977	Aug 5-13		2,4,5-T + 2,4-D	2 lbs/acre a.e.; 3 gals/acre	1990	A0130802
Thunder Bay	Adrian	1974	July 5-15	G	2,4,5-T + 2,4-D	1.81 lbs a.e./acre	65	A0130795
Thunder Bay	Goldie	1971	Aug 4-6	A	2,4,5-T + 2,4-D	2 lbs a.e./acre; 3 gals/acre	414	A0130794
Thunder Bay	Hele	1956	July	A	2,4,5-T + 2,4-D	1.2 lbs a.i./acre 4 gals	500	A0130787, A0132778
Thunder Bay	Wolf River	1977	June 24-26	A	2,4,5-T + 2,4-D	3 gals/acre	1014	A0130801
Tweed		1961	July 21-Aug 10	G	2,4,5-T	2.4 a.e./gallon (1 pint to 4 gals) and 4.8 a.e./gal (2 pints to 4 gals)	152	A0133194, A0133417
Tweed	Ashby, Canton	1979		A	2,4,5-T + 2,4-D		623	A0133112
Tweed	Herschel	1965	Aug 15-31	G	2,4,5-T + DMSO	76.8 oz a.e./gal (3 pints to 45 gals); 10% DMSO by volume	10	A0133170, A0133419
Tweed	Herschel, McClure	1962	May	G	2,4,5-T	76.8 oz a.e./gallon fuel oil; 4% solution	12.7	A0133192

District	Township	Year	Date	Modality[b]	Chemical(s)[c]	Amount used[d]	Acres treated	References[e]
Tweed	Kendall	1957	Aug 21-25	A	2,4,5-T	6 lbs a.e./gallon; 1.5 lbs a.e./4 gals/acre	600	A0133192
Tweed	Radcliffe	1965	Aug 9- Sep 2	G	2,4,5-T		185	A0130589
Tweed	Radcliffe	1966	Aug 1- Sep 6	G	2,4,5-T		185	A0130592
Tweed	Radcliffe	1967	Sep 1- 30		2,4,5-T			A0130596
Tweed	Radcliffe	1968		G	2,4,5-T		425	A0130593
Tweed	Radcliffe	1969	Aug	G	2,4,5-T		240	A0130595
Tweed	Radcliffe	1970	Aug-Sep	G				A0130591
Tweed	Radcliffe	1971	Aug	G	2,4,5-T		85	A0130590
Tweed	Radcliffe	1972	Jul 17-Aug 26	G	2,4,5-T + 2,4-D		280	A0130594
Tweed	Raglan	1966	June 1- Aug 31	G	2,4,5-T		585	A0130584
Tweed	Raglan	1967		G	2,4,5-T		35	A0130587
Tweed	Raglan	1967	Sep 7-14	G	2,4,5-T		50	A0130585
Tweed	Raglan	1970	Aug	G	2,4,5-T + 2,4-D		205	A0130588
Tweed	Raglan	1971	Aug	G	2,4,5-T			A0130586
Wawa	Flood	1968	Aug 4-8	A	2,4,5-T + 2,4-D	2.2 kg/ha; 33.6L/ha		A0129463
Wawa	Hambleton	1970	Aug 13-19	A	2,4,5-T + 2,4-D	33.6 L/ha		A0129459
Wawa	Magone	1971	Aug 10-13	A	2,4,5-T + 2,4-D			A0129457
Wawa	Mikano	1966	July 15- Aug 1	A	2,4,5-T + 2,4-D	45 L/ha; 50/50 mix; 2.77 lbs a.i./acre		A0129454
White River		1966, 1968						A0129548, A0129549
White River		1968	Aug 3-9	A	2,4,5-T + 2,4-D	1:1 mixture ranging in concentration between 31.75 and 34.9 oz acid/acre	2021	A0129549, A0130822
Whitney	Head	1976	Aug 20-23		2,4,5-T		10	A0130601

[a] *Blank cell = no data available.*

[b] *G = ground; A = aerial.*

[c] *2,4,5-T = 2,4,5-trichlorophenoxyacetic acid; 2,4-D = 2,4-dichlorophenoxyacetic acid; TCA = trichloroacetic acid; DMSO = dimethyl sulfoxide.*

[d] *Abbreviations: a.e. = acid equivalent; a.i. = active ingredient.*

[e] *Citations in the format A0 refer to records that are contained within the MNR searchable database. These records are available to the public, subject to Canadian copyright provisions and the Freedom of Information and Protection of Privacy Act. To access these records, visit www.ontario.ca/245T.*

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